EUSTACHIAN TUBE FUNCTION BEFORE AND AFTER SURGERY FOR MUCOSAL CHRONIC SUPPURATIVE OTITIS MEDIA

SUBMITTED BY

Dr. Lisa Mary Cherian

EUSTACHIAN TUBE FUNCTION BEFORE AND AFTER SURGERY FOR MUCOSAL CHRONIC SUPPURATIVE OTITIS MEDIA

A dissertation submitted in partial fulfillment of MS Branch IV, ENT examination of the Tamil Nadu Dr. MGR Medical University, To be held in April 2012.

Christian Medical College, Vellore

Certificate

This is to certify that the dissertation entitled **'Eustachian tube function before and after surgery for mucosal chronic suppurative otitis media'** is the bonafide original work of Dr. Lisa Mary Cherian carried out under my guidance, in fulfillment of the rules and regulations for the MS Branch IV, ENT examination of the Tamil Nadu Dr. MGR Medical University, to be held in April 2012.

Dr. RupaVedantam

Professor and Head

Department of ENT Unit III

Christian Medical College

Vellore.

Acknowledgements

I express my deep sense of gratitude and respect to my esteemed professor and guide Dr. RupaVedantam for taking me on as a thesis candidate and mentoring and guiding me through the process. I am deeply indebted to her support and patience throughout this process and all her encouragement and guidance at every stage of the work on this dissertation.

I would like to thank Dr. Suma Susan Mathew and Dr. Reji Kurien for help and support in carrying out this study

I am grateful to the staff in biostatistics department Ms.Thenmozhi and Mr. Sathiya Murthi and also Ms. Jayasree for patiently analyzing the data and formatting it.

I am extremely thankful to all the audiology lab staff for their constant support

I am indebted to all the patients who consented to be a part of this study and each and every ENT surgeon for the meticulous recording of the findings.

My sincere thanks to all my professors, seniors, colleagues and friends for all their encouragement and help.

A special thanks to my family, especially my husband Dr. Viju Daniel and daughter Tanvi, for supporting me throughout the work on this study.

And above all to the Lord God Almighty for all his blessings.

Table of Contents

AIMS AND OBJECTIVES

1. To assess preoperative Eustachian Tube (ET) function using the inflation deflation technique and Valsalva maneuver in patients with mucosal chronic suppurative otitis media undergoing surgery for perforation closure.

2. To investigate if there is any change in ET function following tympanoplasty in these patients. The null hypothesis is that there is no change in ET function following tympanoplasty

3. To discover whether a patent ET is predictive of postoperative closure of perforation

ABSTRACT

Eustachian tube function before and after surgery in patients with mucosal chronic suppurative otitis media

Introduction

The Eustachian tube (ET), also called the pharyngotympanic tube, is a narrow passage made of bony and cartilaginous parts connecting the middle ear cleft to the nasopharynx. Normal ET function is necessary for ventilation of the middle ear (ME), maintaining the ME space and drainage of ME secretions. Any structural or functional abnormalities of ET can affect these functions and lead to negative ME pressure, fluid accumulation and, eventually, a perforation of the tympanic membrane. The pathogenesis of mucosal chronic suppurative otitis media (CSOM) is thus believed to be linked to abnormal ET function.

Materials and methods

This study was performed in the ENT outpatient department at Christian Medical College Vellore, between May 2010 and July 2011. All patients aged 16 years and above with mucosal chronic suppurative otitis media (CSOM), who were scheduled to undergo surgery, were included in the study. A brief history of their problems and clinical findings, were entered into the proforma .Preoperatively otomicroscopic examination, pure tone audiogram, Valsalva maneuver and inflation-deflation test were done. Post operatively after 3 months, otomicroscopic examination, pure tone audiogram, Valsalva maneuver and tympanometry were done. Of a calculated sample size of 104 (180 patients with 40% loss to follow up), 106 patients were followed up.

Results

Preoperative Valsalva showed that the ET was patent in (54.9%). (62.6%)have blocked ET by the preoperative inflation-deflation test. 72.1% of the patients with a patent ET on Valsalva maneuver showed patent ET on the inflation deflation test also. The association between the 2 tests of ET function preoperatively was high and this was found to be statistically significant ($p<0.01$). When assessed by Valsalva maneuver and inflation-deflation test preoperatively and tympanometry postoperatively, if a patient had a blocked ET preoperatively there was a higher chance of it changing than if a patient had a patent tube ($p<0.001$). The success rate of tympanic membrane closure was 78.4%. Although the patients with non-patent ET had a higher rate of residual perforation, when assessed with Valsalva maneuver and inflation deflation test, there was no statistical correlation between preoperative eustachian tube status and postoperative graft status.

Conclusion

We arrived at 3 important conclusions in this study. Firstly, the ET appeared to be blocked in most patients with mucosal CSOM when assessed preoperatively with the inflation deflation test, whereas most appeared tube patent with the Valsalva maneuver.

Secondly, there was a change in the ET function after surgery which was significant ($p<0.001$) when assessed with both Valsalva and inflation deflation preoperatively and tympanometry post operatively. The function of the ET tends to change from blocked towards patency, possibly due to the removal of the disease and return of normal physiological state after surgery.

Finally, there was an increased tendency of a patient with blocked ET to develop a residual perforation, although this did not reach statistical significance.

KEY WORDS

Eustacchian tube , Valsalva maneuver, Inflation-deflation test.

PRESENT KNOWLEDGE AND REVIEW OF LITERATURE

Introduction

The Eustachian tube (ET), also called the pharyngotympanic tube, is a narrow passage made of bony and cartilaginous parts connecting the middle ear cleft to the nasopharynx. Normal ET function is necessary for ventilation of the middle ear (ME), maintaining the ME space and drainage of ME secretions. Any structural or functional abnormalities of ET can affect these functions and lead to negative ME pressure, fluid accumulation and, eventually, a perforation of the tympanic membrane.

The pathogenesis of mucosal chronic suppurative otitis media (CSOM) is believed to be linked to abnormal ET function.[1] Good preoperative ET function has been associated with high rates of success with regard to tympanic membrane closure.[2] Higher failure rates are seen in patients with poor ET function.[3] Closure of the tympanic membrane perforation may reduce chronic irritation of ME and thus encourage return of normal ET function .[4]

Historical aspects of Eustachian tube anatomy and function

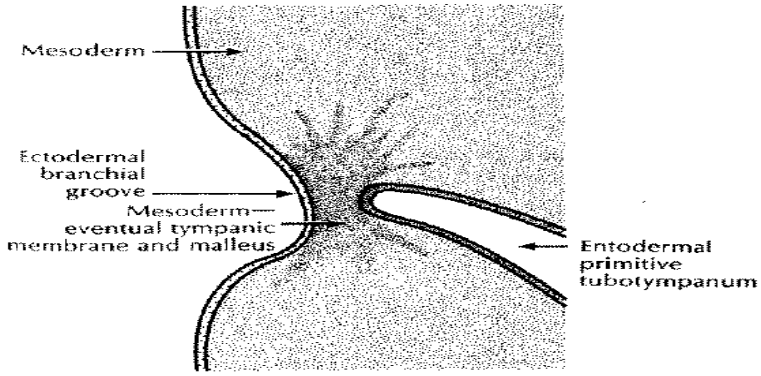
Alcmaeon of Sparta was the first to introduce the term Eustachian tube (ET) in 400 BC.[5] It was Eustachius Bartholomeus (1510 – 1574), who described the ET in his thesis Epistola de auditus organis.[6] Antonio Maria Valsalva(1666 – 1723) described the osseous and cartilaginous part of the ET and identified the 'dilator tubae' part of the tensor veli palati muscle. The Valsalva maneuver which is used to assess the patency of the ET is named after him .[5] Joseph Toynbee (1819 – 1856), a pioneer in aural pathology, described the muscles that open the ET.[7]Adam Politzer,' Father of modern Otology'

introduced the method of Politzerisation, by which air is inflated into the middle ear through the ET which decreases the tension in the middle ear and partly or completely remove secretions thus improving the conductive hearing apparatus.[8]

Embryology

The ET develops from the lateral extension of the endoderm of the first pharyngeal pouch (Figure 1). By the 10th week of intrauterine life the epithelium differentiates, by 12 to 14 weeks the levator veli palati and tensor veli palate develop and the cartilage starts differentiating and epithelial rugae and glandular tissue develops.[9] The length increases from 1mm at 10 weeks to 13mm at birth. The lumen height increases and also the angle between the tensor veli palati and the cartilaginous ET become more acute.[10] As the skull base grows and increases in size, the distance between it and the cartilaginous ET increases. The ET reaches adult size by 7 years of age.[11] The angle between the ET and the Frankfurt horizontal plane changes from 10 degree in infants to 45 degree in adults.[12]

Figure 1: Development of ET and middle ear cleft



Adult Eustachian tube

The ET has an osseous and a cartilaginous part. The length ranges from 31 to 38 mm.[13] The posterior one third (11 to 14 mm) of the adult ET is osseous and the anterior two third (20 to 25 mm) is cartilaginous. The osseous part starts from the middle ear (ME) and travels anteriorly and medially through the petrous temporal bone. The medial wall of this part of ET lies close to the carotid canal and the labyrinth.[14] The osseous ET is lined with epithelium similar to that of the ME and the cartilaginous part with mucosa similar to that of nasopharynx, consisting of ciliated cells and mucous glands.[15] The ET takes a slowly curving inverted 'S' curve from the ME to the nasopharynx.[9] The nasopharyngeal end has an elevation called the torus.

The cartilaginous part has two arms, a short lateral lamina and a longer medial lamina. The lateral arm has a constant height. The medial arm is initially short, and increases to a height of 13mm just behind the attachment of the cartilage to the medial pterygoid plate. Posteriorly the height decreases to 9mm and enters the petrous temporal bone. Thus the dome shaped cartilage continues into the bone between the lumen and the tensor tympani muscle superiorly.[10, 16] The nasal orifice is 8.5mm in height and it gradually decreases posteriorly to 3.5mm. The nasopharyngeal end of the ET is attached to the medial pterygoid and the posterior part to the skull base. The narrowest part of the ET is called the isthmus and it lies in the cartilaginous part. It is around 20mm from the pharyngeal orifice and 0.65mm^2 in cross section. The tensor veli palate tendon is attached to this part in the lateral lamina suggesting that its contraction opens the isthmus to ventilate the ME and also protects the ME.[17]

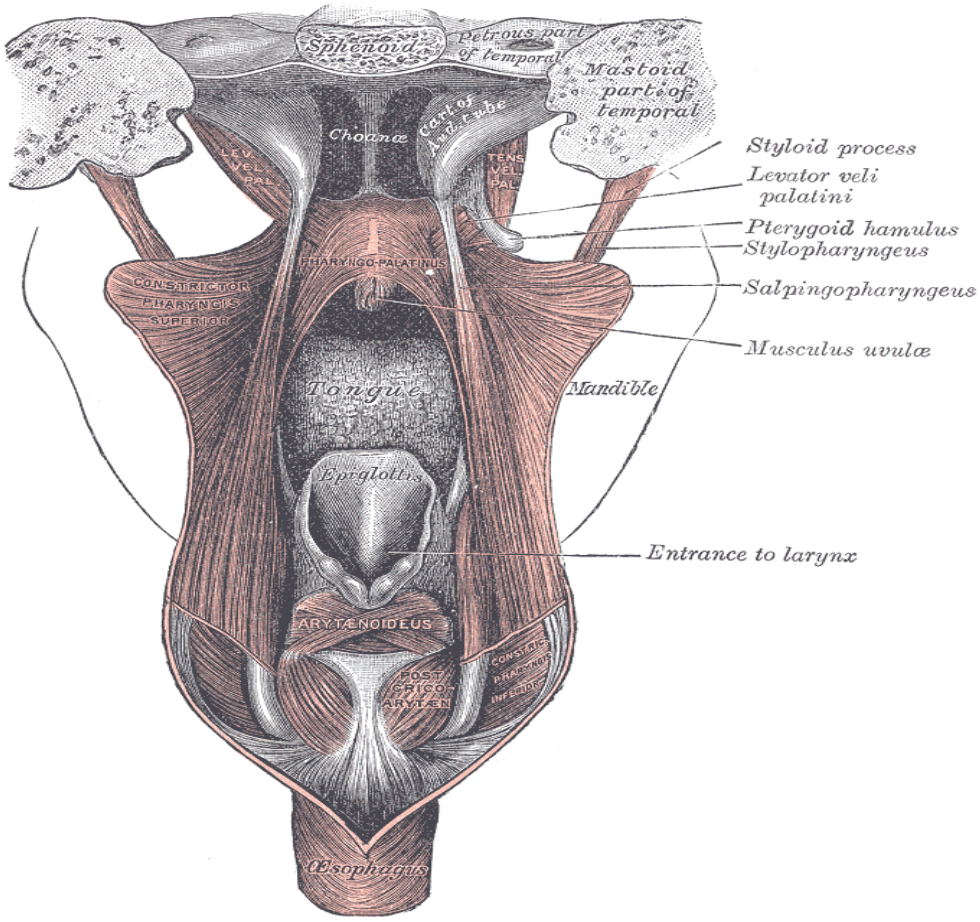
The ET is lined with pseudo stratified ciliated columnar epithelium which is continuous with that of the ME posteriorly and that of the nasopharynx anteriorly. It also contains goblet cells rich in secretory granules, lysozymes and immunoglobulins, A, G, E, and M which contributes to the local immunodefence of the ET.[18] The submucosa contains mucoserous glands which decreases in density with increasing age.[19]

The lateral membranous portion of the ET is defined in the middle portion of the cartilaginous ET. Its medial boundary is submucosa of the lumen. It is anchored superiorly to the inferior curvature of the lateral lamina.[10] The tensor veli palate (TVP) takes its origin from the lateral lamina and the lateral membranous wall.[20]

The muscles associated with ET are: Tensor veli palati, Levator veli palati, salpingopharyngeus, and tensor tympani (Figure-2). The bony part remains open and the cartilaginous ET is closed at rest. It opens during swallowing, sneezing and yawning thus equalising the middle ear pressure to that of the atmospheric pressure. Closure of the tube is achieved by the extrinsic forces of the surrounding deformed tissues and /or by the recoil of the elastic fibres or the tubal wall and cartilage.[10] The TVP consists of two muscle bundles. The more lateral bundle originates from the scaphoid fossa and the greater wing of sphenoid bone superior to the ET cartilage. The muscle descends anteriorly, laterally and inferiorly to converge into a tendon that winds around the hammular process of the medial pterygoid and inserts into the posterior border of horizontal process of the palatine bone and palatine aponeurosis. The more postero-superior muscle fibres extend into the semi canal of tensor tympani muscle and receive a second muscle slip from the tubal cartilage and the sphenoid bone. The fibres converge to a tendon and winds around the processes cochleariformis and inserts into the neck of the malleus. This forms the tensor tympani muscle.[21, 22] The medial bundle originates

from the posterior half of lateral membranous wall of ET. The fibres descend and blend with the lateral bundle. This also called the dilator tubae is the primary dilator of the ET.[21] LVP arises from the inferior aspect of the petrous temporal bone. It lies inferolateral to the inferior margin of the medial lamina of the anterior ET and inferior to the medial lamina of the posterior ET suggesting that the LVP opens the anterior cartilaginous portion of the ET by rotating the medial lamina with increased dimension of the muscle body when it contracts.[23] The Salpingopharyngeus muscle arises from the medial and inferior border of tubal cartilage and passes inferoposteriorly to blend with the Palatopharyngeal muscle. The fibres are very thin and lack ability for any physiological function.[10]

Figure-2: Muscles attached to Eustachian tube.



The middle ear together with the ET, aditus, antrum, and the mastoid air cells is called the middle ear cleft. The ME has a roof, a floor, medial, lateral, anterior and posterior walls. The anterior wall is narrow as the medial and lateral walls converge. The lower portion of the anterior wall consists of a thin plate of bone covering the carotid artery. The smaller upper part has two parallel tunnels placed one above the other. The lower one leads to the bony ET while the upper, separated from the bony ET contains the tensor tympani muscle.

Function of ET

Three physiological functions have been described for the ET.[24] (Figure.3).

1) Ventilation of the middle ear to equilibrate the middle ear pressure with that of the atmosphere. 2) To transport the middle ear secretions to the nasopharyx by mucociliary clearance. 3) To protect the middle ear from nasopharyngeal sound pressure and secretions.

Ventilatory function:

The transducer function (necessary for the normal hearing mechanism) of middle ear is optimal when the ambient and middle ear pressures are equal. This is facilitated by the opening of the ET. The ME is considered as a rigid non-collapsible gas pocket surrounded by vascular mucosa. Gases are exchanged between the middle ear spaces and the mucosa. Partial pressure of gases in the mucosa approximates that of microcirculation of middle ear mucosa. Middle ear O_2 is slightly higher than in microcirculation, whereas CO_2 is in equilibrium.[25] The diffusion gradient is created by N_2 partial pressures and relatively inert gases. When the ET is opened by the paratubal musculature during

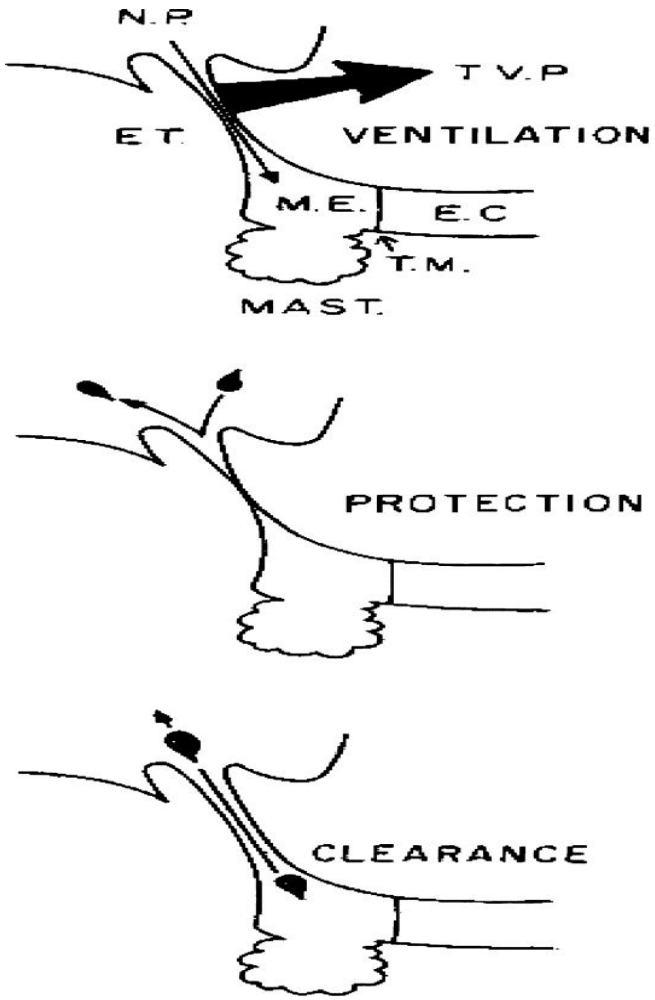
deglutition and other maneuvers, the pressure difference between the environment and the ME are equalised by the inflow and outflow of gases. The ventilator function maintains near equilibrium between internal and external pressures, thus maintaining the near optimal transducer function of the ME and preventing pathological consequences that result from unabated ME to mucosa gas exchange. In an attempt to describe normal ET function using micro flow technique in a pressure chamber, Elner et al[26] studied 102 adults with intact tympanic membrane and no ear disease. The patients were divided into four groups according to their ability to equilibrate static relative positive and negative pressures of 100mm of water in the ME. Group 1 equilibrated the pressure completely; group 2 equilibrated positive pressure, but had a small residual negative pressure in the ME. Group 3 were capable of equilibrating only relative positive pressure with a small residual remaining, but not negative pressure; those in group 4 were unable to equilibrate any pressures. This indicated decreased stiffness of ET in group 2 to 4 when compared to group 1. This study also showed that 95% of normal adults could equilibrate an applied positive pressure and 93% could equilibrate applied negative pressures to some extent by active swallowing. However 28% of the subjects could not completely equilibrate either applied positive or negative pressure or both.

Protection, drainage and clearance

The ET, ME and mastoid air cells can be considered as a flask with a long narrow neck.[27] The nasopharyngeal end can be considered as the mouth of the flask, the narrow neck, the isthmus of the ET and the bulbous portion the ME and the mastoid air cells. Fluid flow through the neck will depend upon the pressures at both ends, the radius and length of the neck and the viscosity of the fluid. When a small amount of fluid is instilled into the mouth of the flask, fluid stops somewhere at the narrow neck as a result of

capillarity at the neck and positive pressure that develops in the chamber of the flask. If there is a perforation of the tympanic membrane, secretions can move from the nasopharynx to the ME as there is no positive pressure from the mastoid to prevent the reflux. If a high negative pressure develops in the ME and the mastoid, fluid gets aspirated into the ME and mastoid from the nasopharynx. Similarly if a high positive pressure is applied at the nasopharynx, fluid insufflates into the ME. The other factors which affect the flow of gas and fluid through the ET are 1) mucociliary clearance.[28, 29], 2) active tubal opening and closure, 3) Surface tension characteristics.[30]

Figure.3 Functions of ET (ventilation, protection and clearance)



Pathophysiology of middle ear disease

There are various factors associated with the etiology and pathogenesis of ME disease. These include ET dysfunction, other host related factors like genetic predisposition, age, prematurity, allery, immunodeficiency, craniofacial abnormalities, environmental factors like upper respiratory tract infections, attendance to day care, number of siblings, absence of breast feeding, and socioeconomic factors. ET dysfunction is one of the most common causes of ME diseases. There are many studies which show sufficient evidence to support the 'hydrops ex vacuo' theory of Adam Politzer, which says that when the ET is blocked and there is edema of the ME mucosa, ME under pressure develops and there is transudation of serous fluid into the ME.[31]Anatomic or functional obstruction of the ET is the primary cause of ME negative pressure. Anatomic obstruction is frequently due to inflammation mostly due to viral infection. Others are inflammation due to allergy or obstruction at the nasopharyngeal end due to adenoids or tumour and the middle ear due to granulations. Functional causes are floppy cartilage, dysfunction of the TVP muscle, constriction of the ET while swallowing and otic barotrauma. Secondary cause of ME negative pressure is inflammation in the ME. This can lead to obstruction of the bony ET with secretions, thus impairing the pressure regulatory and clearance functions of the ET. ME under pressure leads to acute otitis media (Figure.4) and otitis media with effusion (Figure.5). Acute otitis media with otorrhea occurs when there is rupture of tympanic membrane. If acute otitis media with tympanic membrane rupture does not heal spontaneously or following treatment it can lead to chronic otitis media (Figure.6). Negative ME pressure also enhances reflux and insufflations of secretions from nasopharynx to ME. Otitis media can also occur in

dysfunction of the ET due to reflux or insufflation of nasopharyngeal secretions into ME, which can occur in condition like patulous ET or cleft palate. In chronic suppurative otitis media the ME and mastoid get infected from the nasopharyngeal secretions and the contaminants from the external auditory canal (Figure.7). One of the most important factors associated with otitis media in infants and young children is immaturity of the structure and function of the ET and immaturity of the immune system.[32]

The antecedent event in an acute otitis media is a viral upper respiratory tract infection. This leads to congestion and inflammation of the mucosa of the nasopharynx and ET and thus obstruction of the ET. Further development of ME under pressure occurs especially in mastoids, where the mastoid air cell system (MACS) volume is less. Larger MACS will partially protect the ME from development of a pathological under pressures by allowing for a decreased frequency of ET openings and/or less efficient ET openings when compared to a smaller MACS volume.[33] Negative ME pressure is followed by aspiration of pathogens (viral and bacterial) into the ME from the nasopharynx. This along with the secretions from the middle ear mucosa gets collected, allowing the pathogens to proliferate that eventually leads to suppuration. In a study by Buchman[34] and colleagues where 32 healthy adults were subjected to a nasal inoculation of respiratory syncytial virus, 18 (56%) of them were infected with the virus and by the 6^{th} day only 46% of the infected subject had a normal ME pressure (> 100mmof H_2O), showing the relationship between viral upper respiratory tract infection and otitis media.

Otitis media with effusion has a similar sequence of events as that of acute otitis media. Swarts[35] and associates were also able to produce ME effusion in the monkey shortly after inducing negative middle-ear pressure by flushing the middle ear with CO_2. This study confirm the 'hydrops ex vacuo' theory of the pathogenesis of ME effusion, which postulates that in the absence of ET opening, the gas exchange from the middle ear

into the microcirculation of the mucous membrane causes a middle-ear under pressure, followed by transudation of effusion.

Tympanic membrane perforations that are acute are usually secondary to AOM which may also occur during the course of chronic otitis media with effusion .[36] Chronic perforation of the tympanic membrane usually occurs when an acute perforation fails to heal. When there is acute drainage through perforated tympanic membrane that persists for 2 weeks to 3 months or longer the infection is chronic suppurative otitis media. There is no consensus on the duration of otorrhea to be termed chronic; however, as per the WHO definition.[37], otorrhoea persisting through a perforated tympanic membrane for > 2 weeks is believed to be due to CSOM. It has been shown that in patients with chronic otitis media with effusion there is degeneration of lamina propria of the tympanic membrane with decrease in thickness of fibrous layers.[38] This weakness of tympanic membrane predispose to perforation with less chance of spontaneous healing. Thus the pathogenesis of CSOM is linked to abnormal ET function.[32] In inactive mucosal CSOM, the structure and often the hearing is impaired by the presence of a permanent tympanic membrane defect but there is no active infection or mucus discharge. Such an ear may remain inactive, become active or may even occasionally heal. Active mucosal CSOM is associated with resorption of parts or all of the ossicular chain (resoptive osteitis).[39] (Figure.8).

Figure.4: Acute suppurative otitis media

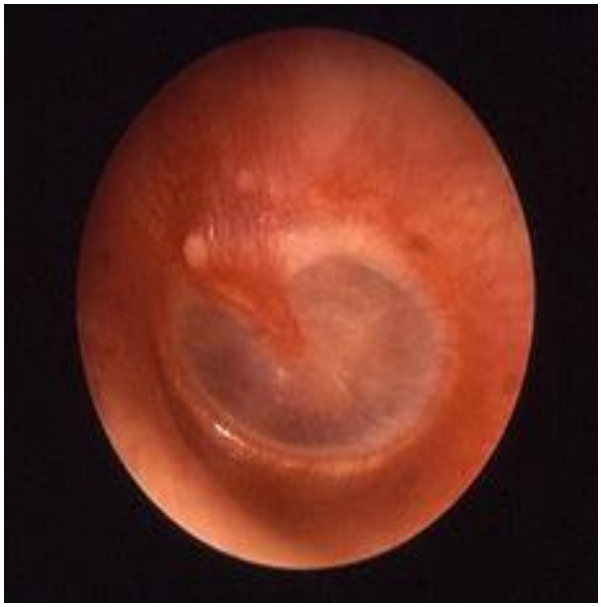


Figure.5: Otitis media with effusion

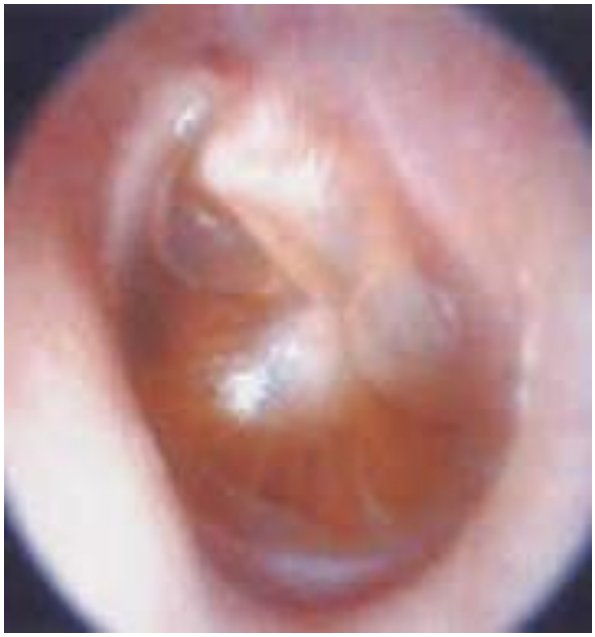


Figure.6: Chronic suppurative otitis media - tubotympanic disease

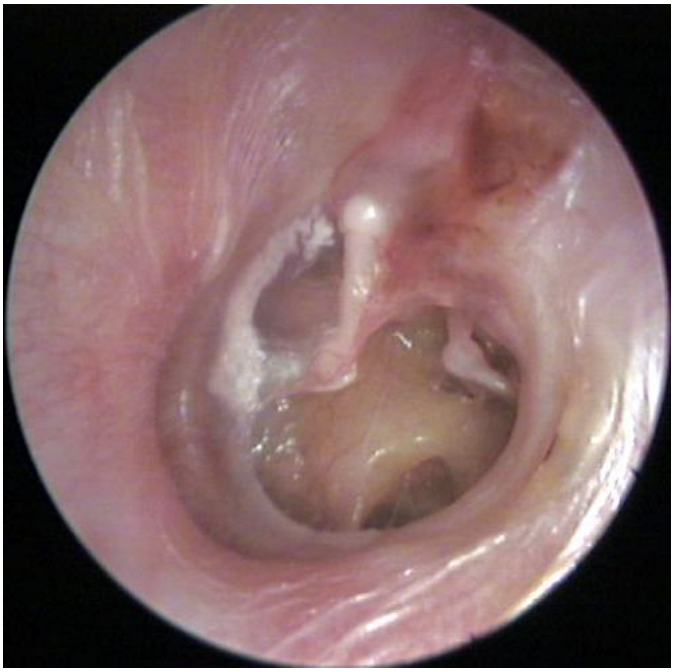


Figure.7 Infected fluid in the middle ear

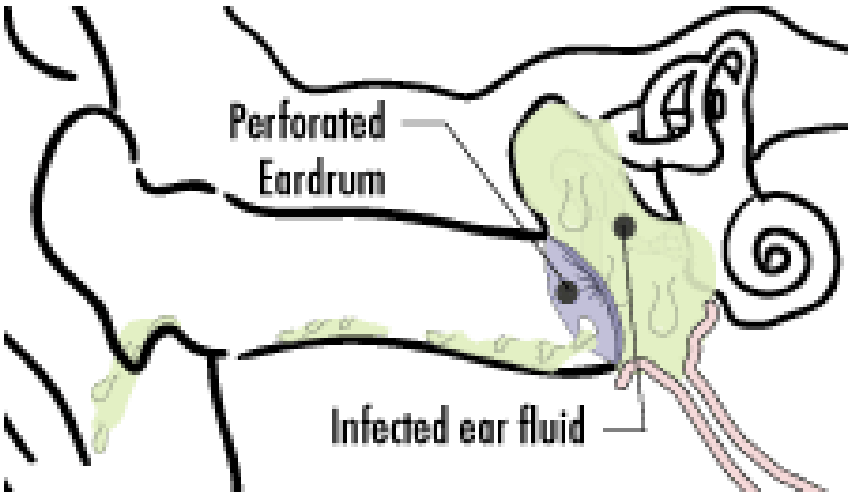
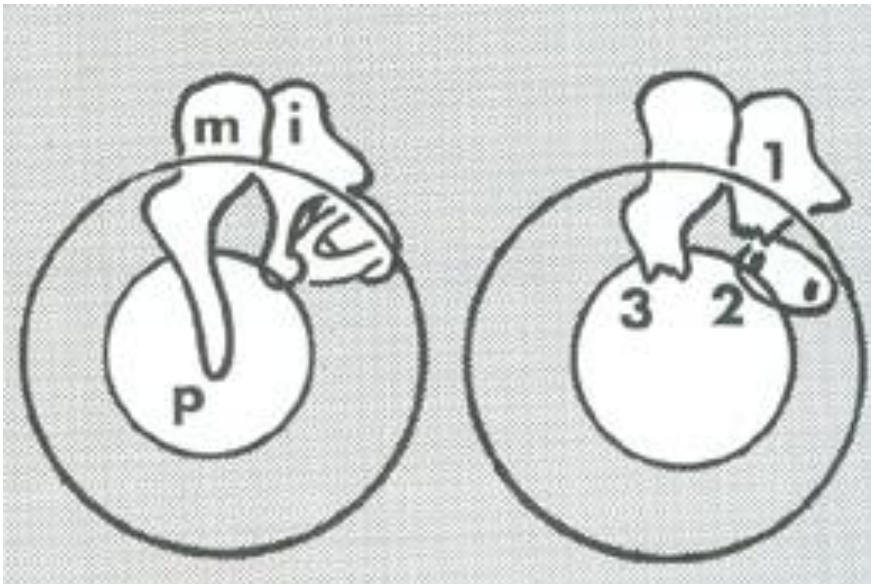


Figure.8:Resorptive osteitis



Surgical treatment of CSOM tubotympanic disease

The main objectives of surgery in patients with CSOM tubotympanic disease (TTD) are

1) To remove the active disease and promote healing

2) To restore an air filled ME cavity and prevent recurrence.

3) To prevent complications

4) To restore hearing function

The surgical procedures often done are myringoplasty, tympanoplasty and cortical mastoidectomy with tympanoplasty. Myringoplasty (Figure.9) involves excision of the

edges of the perforation and placement of a graft of temporalis fascia or perichondrium by an underlay or overlay technique to close the perforation. Tympanoplasty involves reconstruction of tympanic membrane and/or the ossicular chain. Studies on outcomes in terms of tympanic membrane uptake in these surgeries have been reported to be 78% and 95% in the hands of trainees and senior staff respectively.[40] Many authors suggest that a cortical mastoidectomy (Figure.10) should be carried out at the same time as myringoplasty in active ears and inactive ears.[41] In a study by Mishiro et al[42] in patients with mucosal CSOM, 251 ears were operated and followed up for one year. It was divided into two groups; group A with 147 patients underwent tympanoplasty with mastoidectomy and group B with 104 tympanoplasty without mastoidectomy. Graft success rates were 90.5% in group A and 93.3% in group B. There was no statistically significant difference in graft success rate. Graft success rates of discharging ears were 90.0% in group A and 85.7% in group B. Graft success rates of dry ears were 90.7% in group A and 94.4% in group B. There was no statistically significant difference between discharging ears and dry ears. The rates of the postoperative air-bone gap within 20dB were 81.6% in group A and 90.4% in group B, without a statistically significant difference. This study shows that mastotoidectomy need not be combined with tympanoplasty for non cholesteatomatous CSOM, even if the ear is discharging.

Figure.9: Myringoplasty

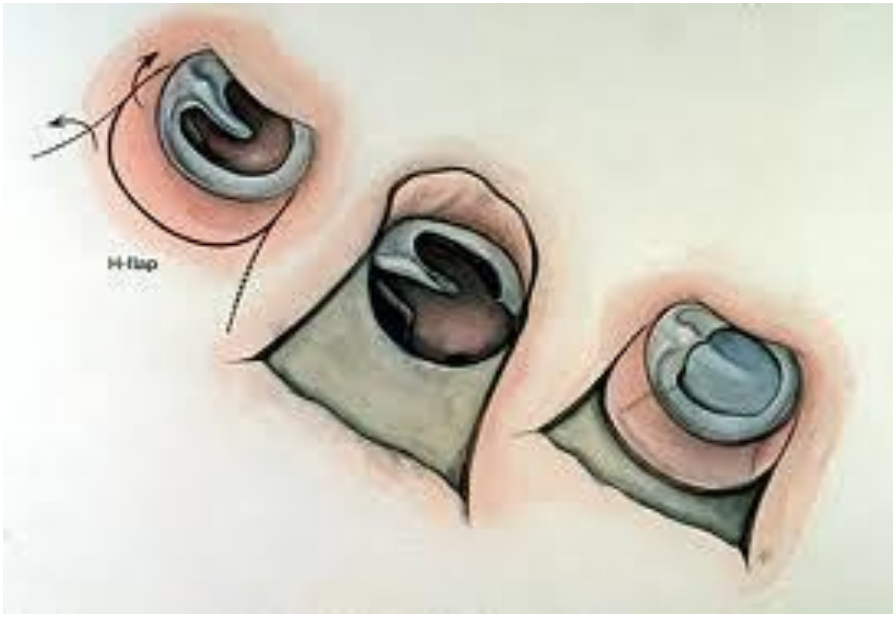
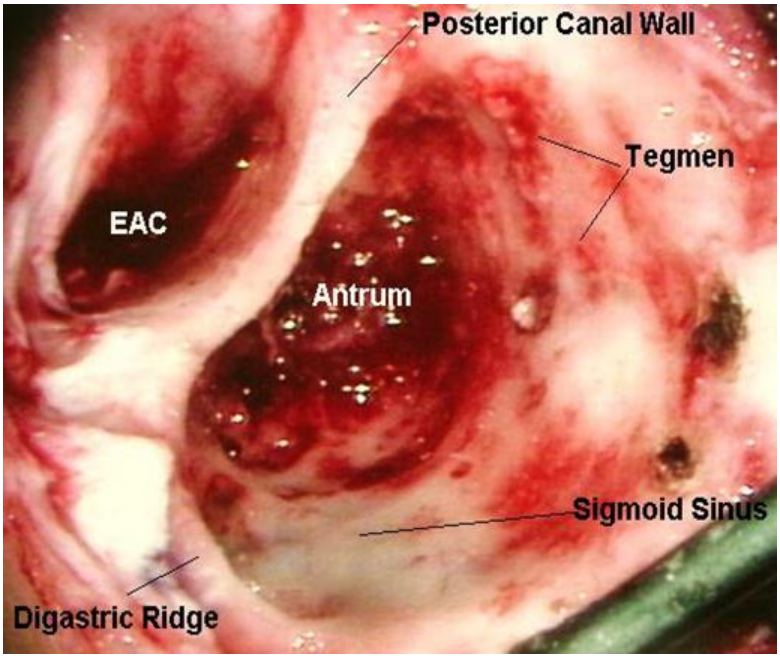


Figure.10: Cortical mastoidectomy

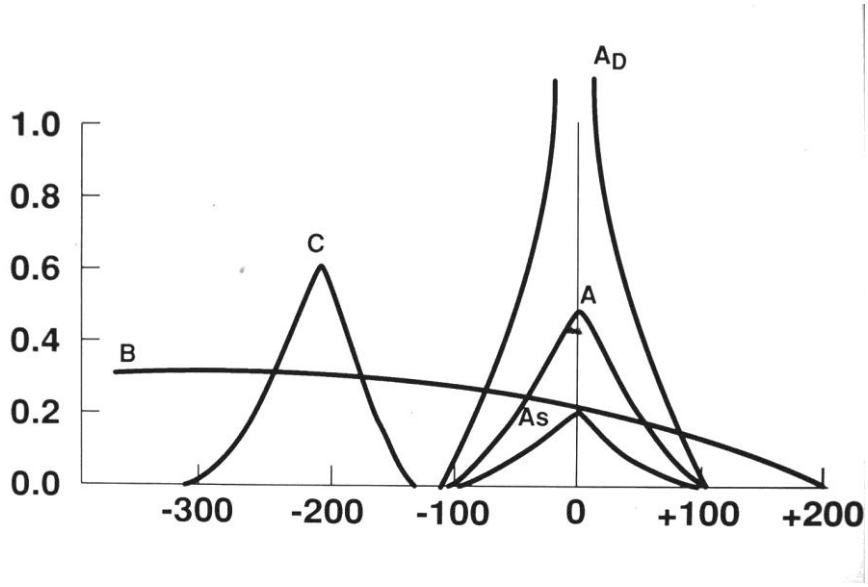


Eustachian tube function tests

There are various tests to assess the function of the ET. These include pneumatic otoscopy.[43], nasopharyngoscopy and ET endoscopy[44] and tympanometry. The latter is an objective way of determining ME pressure and thus the function. It is used to test the condition of the middle ear and mobility of the tympanic membrane by creating variations of air pressure in the ear canal. A compliance peak on a tympanogram indicates that the ear canal and middle ear pressures are equal, and that acoustic transmission through the middle ear is maximal. Tympanograms (Figure.11) are classified with respect to this peak.[45] Some of the classical tests of tubal patency include Valsalva maneuver, Toynbee test, ET catheterization and Politzerisation. When the tympanic membrane is intact and

the ME inflates following Valsalva or Politserisation, then the tube is not totally obstructed. Similarly if the tympanic membrane is not intact, the passage of air into the ME indicates ET patency. Elner et al[26] reported that 86% of otologically normal adults could perform Valsalva. The other tests include dye studies with fluroscien.[46], saccharine.[47] and sonotubometry.[48]

Figure.11: Tympanogram



Drawbacks of the ET function tests:

Otoscopy cannot differentiate between a functional impairment and mechanical obstruction of the ET. Valsalva maneuver is an objective method of assessment of ET function and is difficult to perform in the paediatric age group. Catheterization can traumatise the ET while inserting the catheter. Tympanometry which is done in an intact tympanic membrane gives us only relative qualitative information of the tubal function.

Figure 12: Tympanometer – used for measuring inflation-deflation test.

Inflation-deflation test was first reported by Flisberg et al.[49] It can be done in ears with chronic suppurative otitis media with a perforated drum, in patients who have undergone myringotomy and grommet insertion for otitis media with effusion and also in an intact tympanic membrane. The test is easy to perform, non-invasive and reproducible. It is possible by this method to determine at which level of negative or positive pressure applied within the ear space air passes through the tube during swallowing. (Figure.13, 14)

Figure.13: Inflation test of Eustachian tube function

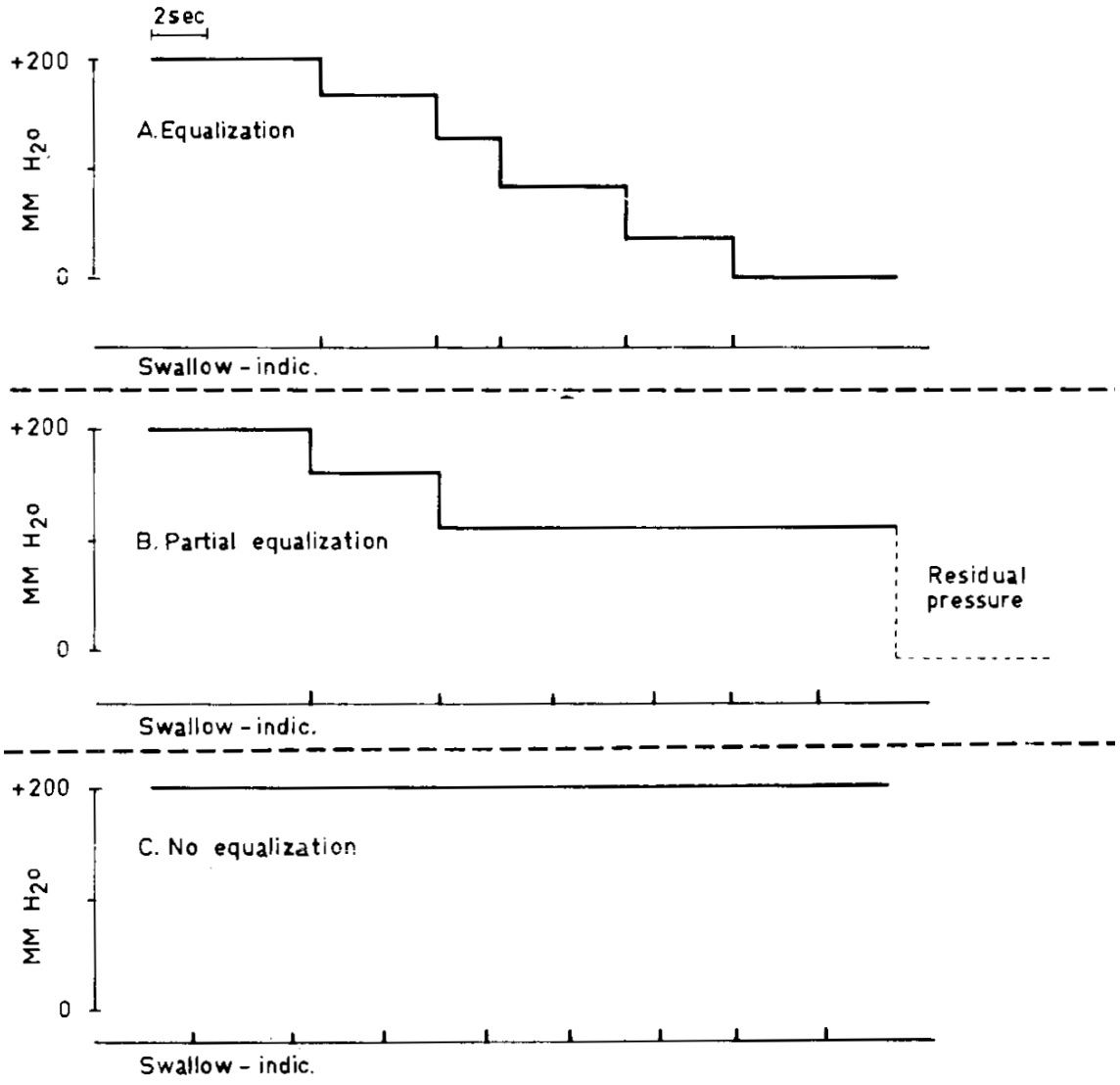
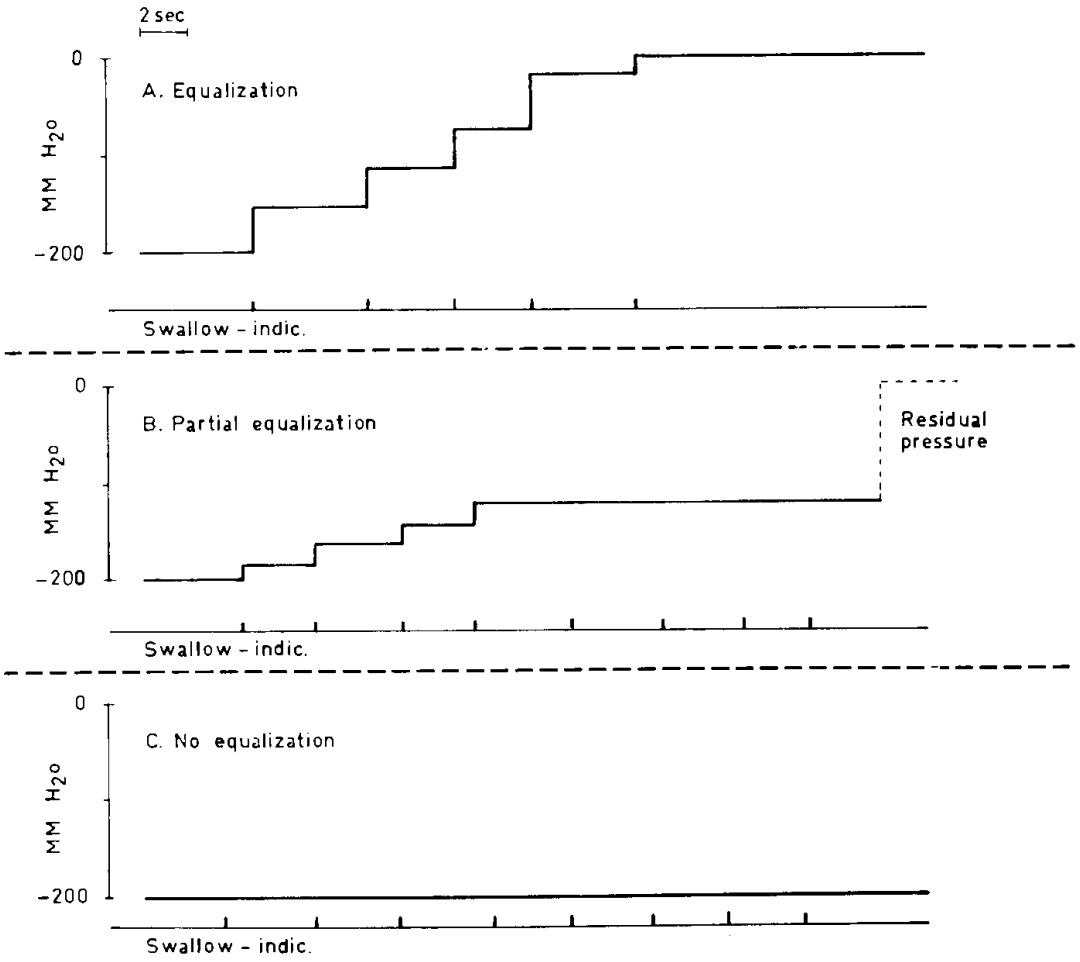


Figure.14: Deflation test of Eustachian tube function



Takahashi et al[50] reviewed 78 patients with non cholesteatomatous chronic otitis media, without ossicular damage. ET pressure-regulation functions were examined using an inflation-deflation test, and postoperative hearing levels were measured. Patients with poor pressure regulation showed a significantly higher incidence of poor outcomes, poor hearing recovery, spontaneous perforation of the tympanic membrane, or persistent wet condition. Uzun[51] reported 61 ears with pars tensa cholesteatoma who underwent tympanoplasty with cartilage palisades or fascia. The authors evaluated preoperative ET function using the Valsalva maneuver and found that late hearing results were better in patients with preoperative positive Valsalva maneuver than they were in those with negative maneuver. With respect to ears with poor tubal function, hearing results were

significantly better in those treated with cartilage palisade tympanoplasty than they were in those treated with fascia reconstruction. The authors suggested that cartilage palisade tympanoplasty might be a better reconstruction option for patients with poor ET function. Choi et al[52] in their study of in their study of 137 patients with non cholesteatomatous ears found that a successful type 1 tympanoplasty and restoration of well aerated ME and good ET function permit better compliance of tympanic membrane and predictive of improved postoperative hearing. According to a study done by Kurien et al[53], the ET inflation deflation test is a sensitive test for predicting aditus patency in patients with dry ear and hence could be used to avoid unnecessary mastoid surgery

MATERIALS AND METHODS

This study was performed in the ENT outpatient department at Christian Medical College Vellore, between May 2010 and July 2011. All patients aged 16 years and above with mucosal chronic suppurative otitis media (CSOM), who were scheduled to undergo surgery, were included in the study. A brief history of their problems and clinical findings, were entered into the proforma. Otomicroscopic examination was performed to assess the size and site of the tympanic membrane perforation. The presence of tympanosclerosis, granulations, discharge and exposure of round window or ossicles was noted. Details were entered into the proforma.

Exclusion criteria: Presence of any of the following conditions:

- Profusely discharging ear
- Cholesteatoma
- Immunosuppression
- Poorly controlled diabetes mellitus

Methods:

a) Audiological assessment

Hearing assessment was performed by pure tone audiometry to note the mean value of the air-bone gap at 500, 1000, 2000 and 3000 Hz. Hearing loss was quantified as per Goodman's scale as follows:

Mild	-	26-40 dB
Moderate	-	41-55 dB
Moderately severe	-	56-70 dB
Severe	-	71-90 dB
Profound	-	> 90 dB

b) ET patency was assessed on the day prior to surgery by the following method:

1. Valsalva maneuver

In this maneuver positive pressure is built in the nasopharynx by sealing it at the nasal and pharyngeal openings so that air enters the ET. This was done by asking the patient to pinch his nose and close his mouth and blow air into his ears. ET was deemed to be patent if the patient got a subjective sensation of opening of the ear or air passing through the ear. This test was done both pre and postoperatively.

Inflation-deflation test

The external auditory canal of the patient was fitted with a rubber ear tip of corresponding size so as to maintain an air tight seal. The otoadmittance probe was connected to a standard otoadmittance meter (GSI Ltd). The middle ear was then inflated with a constant flow of air until the ET is opened passively. This was marked by a sudden fall in pressure (opening pressure). This was the forced response test.

The middle ear pressure was then equilibrated by performing 5 consecutive swallows with an interval of ten seconds in between to prevent strain on the pharyngeal muscles (active tubal function). The residual pressure after 5 swallows if any was recorded. The deflation phase of the test was performed by giving a pressure of - 400 mm to the middle ear and asking the patient to swallow again. The pressure remaining in the middle ear after 5 consecutive swallows was recorded. The ET was considered to be patent if there was no or minimal residual pressure during both inflation and deflation.

The results of the inflation deflation test were categorised based on whether the patients could equalise pressure or have a residual pressure of 100mmof H_2O or less as being patent. Those patients who could not equalise pressures on swallowing either with positive or negative pressures to <100mm of H_2O were deemed to have a non-patent or blocked ET. This group included patients in whom there was no change with either positive or negative pressure.[54] The participants of the study underwent myringoplasty or tympanoplasty with cortical mastoidectomy on the following day. The tympanic membrane perforation was closed using a temporalis fascia graft.

c) Postoperative assessment

1. Otomicroscopy was performed to note the integrity of the tympanic membrane. The following specific features were observed with respect to the tympanic membrane:

    i. 1 – Normal

    ii. 2 – Perforation

iii. 3 – Retraction

iv. 4 - Tympanosclerosis

v. 5 – Granulation over graft

2. Postoperative pure tone audiometry was performed at the same frequencies as preoperatively.

3. Postoperative middle ear aeration was assessed in those patients with intact tympanic membranes using tympanometry. An automated otoadmittance meter was used to record a tympanogram. The results of tympanometry were expressed as follows:

    A-normal

    As-Reduced compliance at ambient pressure

    Ad-Increased compliance at ambient pressure

    B-flat or dome shaped

    C-Maximum compliance at pressures more than -100 mm H_2O (Blocked ET tube)

Sample size calculation:

Sample size for this study was calculated using the following formula

$$n = \frac{(Z_{1-\alpha/2} + Z_{1-\beta})^2 * 2 * PQ}{(p_1 - p_2)^2}$$

Where,

n = sample size

p_1 = incidence of blocked ET postoperatively in patients with patent ET preoperatively

p_2 = incidence of blocked ET postoperatively in patients with blocked ET preoperatively

$P = (p_1 + p_2) / 2$

$Q = 1 - P$

Alfa = 5%

Beta = 80%

This calculation was derived based on the study by Choi HS et al[52] in which poor ET function was seen postoperatively in 40% of those patients with CSOM who had minimal or no residual pressure (good ET function) preoperatively and 67% of those who had moderate to large amounts of residual pressure (poor ET function) preoperatively. Using these figures we estimated that in order to detect this difference at 5% level of significance, with a power of 80% the required sample size is 57 in each group. With a speculated loss to follow up of 40%, the number required in each group will be 90 subjects (total of 180 subjects).

Statistical analysis

Descriptive analysis was used for frequency calculation. This was used to analyse the preoperative and postoperative ET function. Pearson Chi-square test and Fisher's exact test was used for categorical data to check the association. The change in ET function and the association of the ET function with graft uptake were analysed using Pearson Chi-square test.

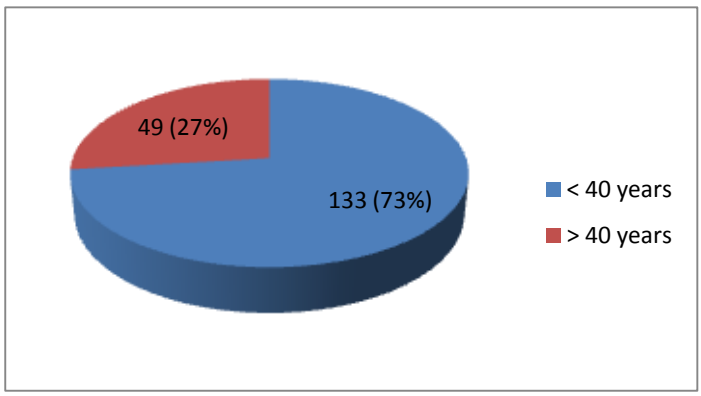
RESULTS AND ANALYSIS

Of the 182 patients in our study who underwent surgical closure of tympanic membrane, 106 of them were reviewed postoperatively over a period of 3 to 15 months.

Table 1: Distribution of age of study patients (n = 182)

Age (yrs)	Number (%)
<40	133 (74.1)
>40	49 (26.9)
Total	182

Figure 15: Age distribution of study patients

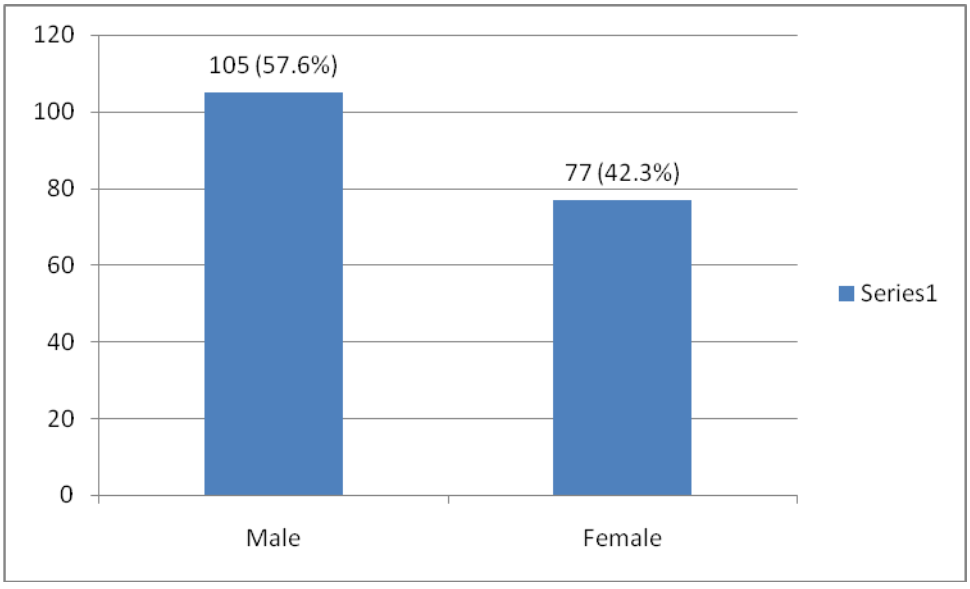


The age range was 16-59 years with a mean age of 32.6 years. Most patients (74.1%) of them were below the age of 40 years or less. This was probably because it was patients in this age group who chiefly sought surgery for ear discharge and hearing loss.

Table 2: Distribution of sex (n = 182)

Sex	Number (%)
Male	105 (57.6)
Female	77 (42.3)
Total	182

Figure.16: Sex distribution of study patients



There were more males than females.

Table 3: Laterality of disease in affected patients (n = 182)

Side	Number (%)
Unilateral	102 (56)
Bilateral	80 (44)
Total	182

Unilateral disease (56%) was slightly more common than bilateral disease (44%).

Figure 17: Distribution of laterality of ear disease:

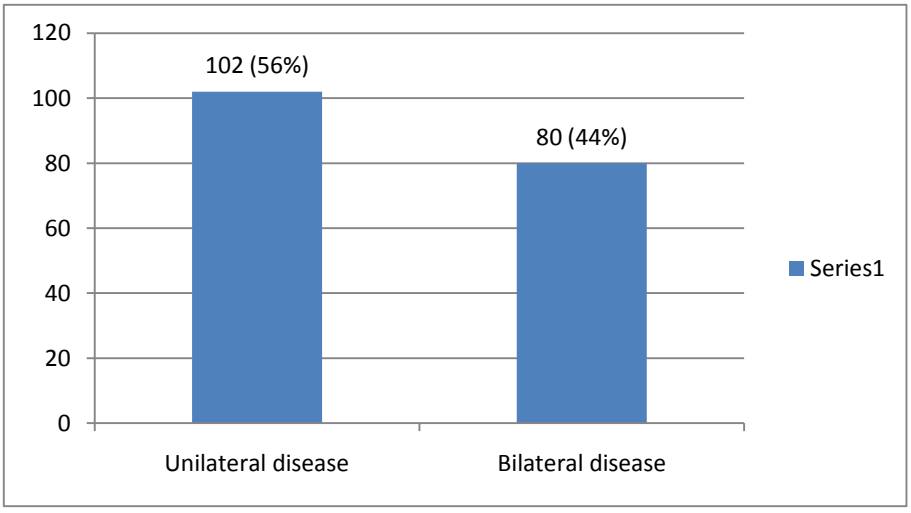


Table 4: Distribution of duration of ear discharge (n=182)

Duration of ear discharge	Number (%)
< 10yrs	107 (58.8)
>10yrs	75 (41.2)
Total	182

The duration of the ear discharge ranged from (0.08 yrs to 50 yrs). While most patients had ear discharge for less than 10 years, a significant number (41.2%) had discharge of > 10 year's duration. Of these, only a small proportion had sought surgery.

Figure 18: Distribution of duration of ear discharge

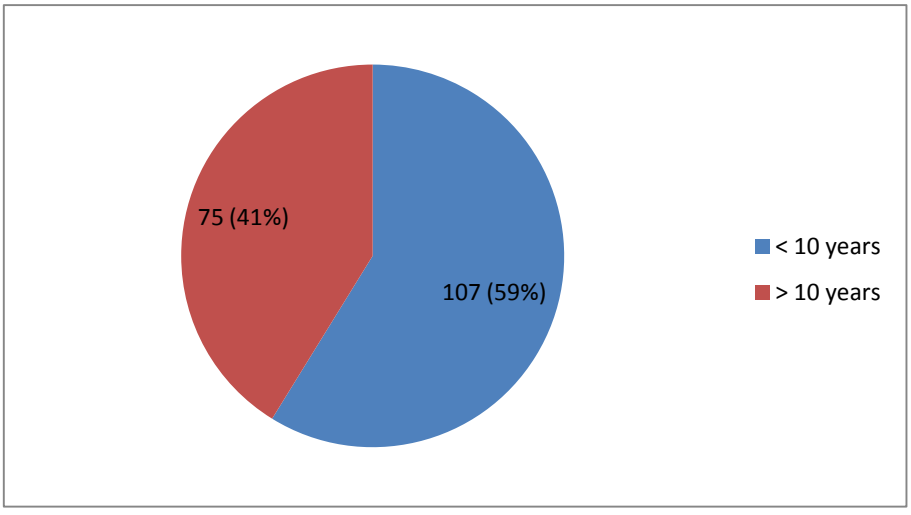


Table 5: Distribution of revision surgery (n = 182)

Previous surgery	Number (%)
Revision surgery	14 (7.6)
First surgery	168 (92.3)
Total	182

Most of the ears (92.3%) were operated for the first time and only 7.6% had undergone revision surgery.

Figure 19: Distribution of revision surgery

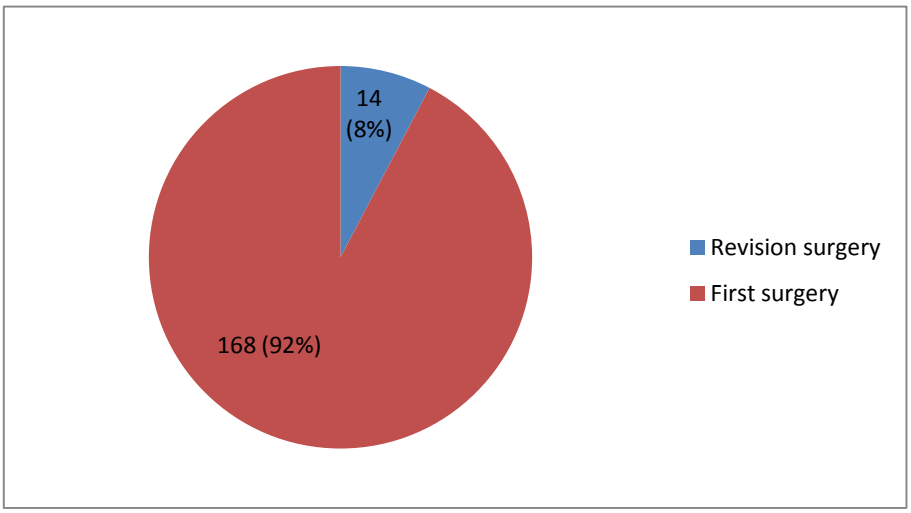


Table 6: Distribution of co morbidities (n = 182)

Comorbid conditions	Number (%)
Present	21 (11.5)
Absent	161 (88.5)
Total	182

11.5% of the patients had comorbid conditions like diabetes mellitus, hypertension, hypothyroidism or bronchial asthma.

Figure 20: Distribution of comorbid conditions

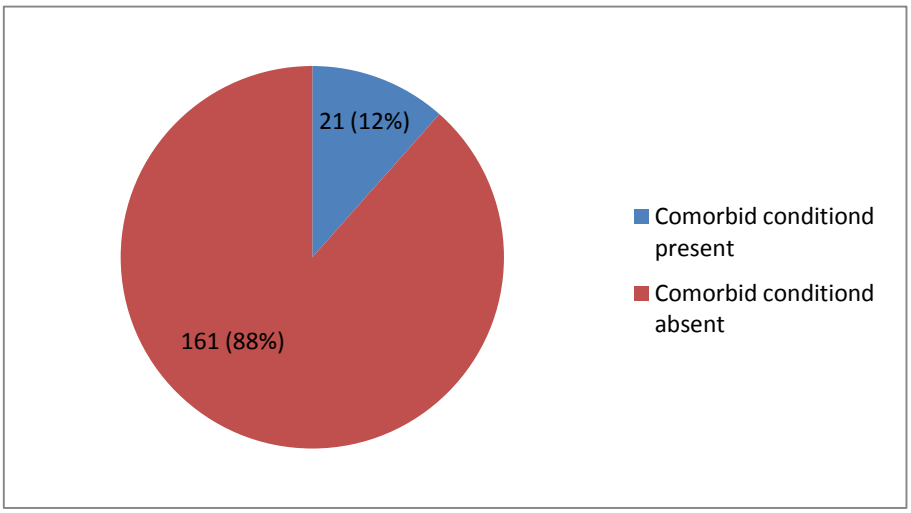


Table 7: Distribution of perforation size (n = 182)

Size of perforation	Number (%)
Small / Moderate	36 (19.8)
Large / Subtotal	146 (80.2)
Total	182

Most (79.6%) of the patients had a large or subtotal sized perforation.

Table 8: Distribution of state of middle ear mucosa (n = 182)

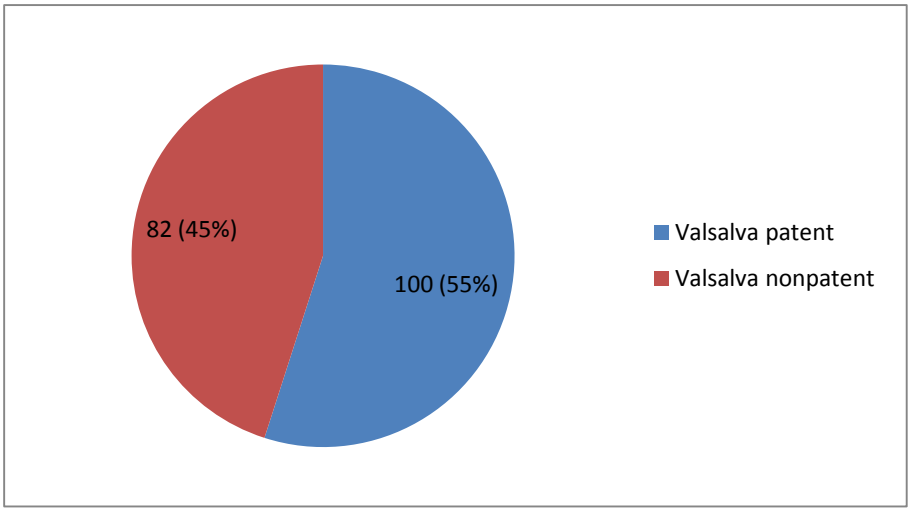
Middle ear mucosa	Number (%)
Dry	92 (50.5)
Moist	90 (49.5)
Total	182

Dry and moist perforations were almost equally distributed.

Table 9: Distribution of preoperative Valsalva maneuver test results (n = 182)

Preoperative Valsalva maneuver test results	Number (%)
Patent	100 (54.9)
Not patent	82 (45)

Figure.21: Distribution of preoperative Valsalva maneuver

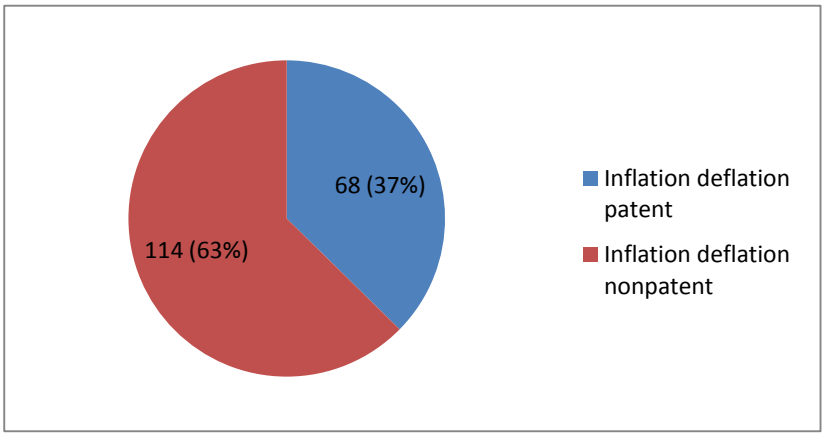


Preoperative Valsalva showed that the ET was patent in a little over half of the patients. Thus, this test of ET function showed that ET dysfunction was not an important cause of the disease in this group of patients.

Table 10: Distribution of preoperative inflation and deflation test results (n = 182)

Preoperative inflation & deflation test results	Number (%)
Patent	68 (37.4)
Not patent	114 (62.6)
Total	182

Figure.22: Distribution of preoperative inflation deflation test



The other test of ET functioned employed in this study was the inflation-deflation test. The test was done in patients with mucosal chronic suppurative otitis media preoperatively. Most patients appeared to have blocked ET by the preoperative inflation-deflation test. The results of this test are quite different from that of the Valsalva test which showed that a little over half the patients had patent ET.

Table 11: Distribution of antral contents (n =114)

Antral mucosa	**Number (%)**
Normal mucosa	57 (50)
edematous mucosa	57 (50)
Total	114

Almost equal numbers of patients had either edematous or normal mucosa.

Table 12: Distribution of aditus status (n =114)

Aditus status	Number
Patent	57(50)
Blocked	57(50)
Total	114

Equal numbers of patients had blocked and patent aditus intra operatively. This is probably related to the status of the antral mucosa. Thus patients with edematous mucosa would probably have a blocked aditus.

Table 13: Association between preoperative Inflation deflation test result and preoperative Valsalva result

Preoperative Valsalva test result	I-D patent	I-D not patent	Test used for significance	P value
Patent	49 (72.1%)	51 (44.7%)	Pearson Chi-square without Yates correction	0.000*
Not patent	19 27.9%)	63(55.3%)		

72.1% of the patients with a patent ET on Valsalva maneuver showed patent ET on the inflation deflation test also. The association between the 2 tests of ET function preoperatively was high and this was found to be statistically significant ($p<0.01$).

Postoperative follow up was obtained in 106 patients, all of whom underwent tympanometry and otoscopy.

Table 14: Distribution of postoperative results (n = 106)

Postoperative tympanic membrane status	**Number (%)**
Intact tympanic membrane	83 (78.4)
Residual perforation	23 (21.6)
Total	106

There were a total of 106 patients who came back for follow up. The success rate of tympanic membrane closure was 78.4%. 21.6% had a residual perforation.

Table 15: Distribution of postoperative tympanometry test results (n =83)

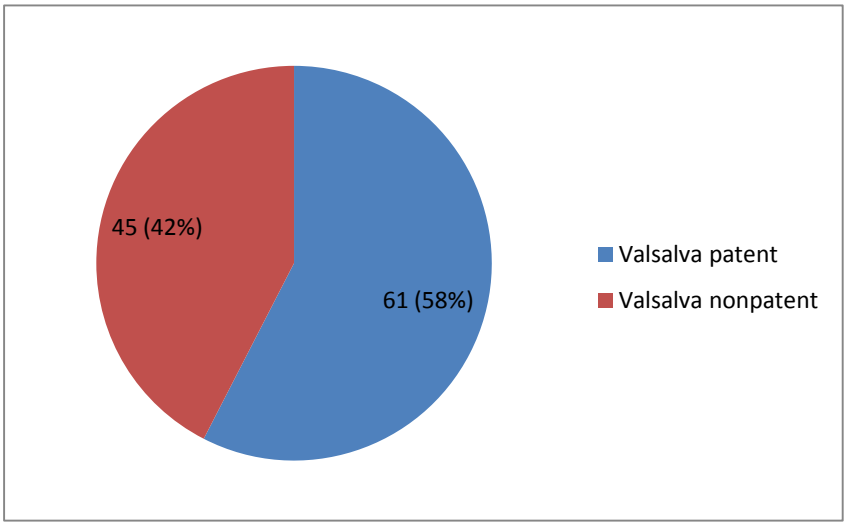
Postoperative tympanometry test results	Number (%)
A, As, Ad	60 (72.2)
C, Cs, B	23 (27.7)
Total	83

Postoperative tympanometry was done to assess the middle ear status and the ET function in all those with intact tympanic membranes. 72.2% had a normal postoperative tympanometry and 27.7% had an abnormal tympanometry. The presence of B curves suggests either the presence of middle ear fluid or thickened tympanic membrane, those with C curves probably had ET block.

Table 16: Distribution of postoperative Valsalva test results (n = 106)

Postoperative Valsalva maneuver test results	Number (%)
Patent	61 (57.5)
Not patent	45 (42.5)
Total	106

Figure 23: Distribution of postoperative Valsalva maneuver



There was an increase in the number of patients with postoperatively patent ET (57.5%) when performing the Valsalva maneuver.

Table 17: Comparing the change in ET function assessed by Valsalva maneuver preoperatively and Valsalva maneuver postoperatively

Preoperative Valsalva maneuver test result	Change present in ET function assessed by Valsalva maneuver (%)	No change in ET function assessed by Valsalva maneuver (%)	Test used for significance	P value
Patent	12(48)	48(59.2)	Pearson Chi-square without Yates correction	0.320
Not patent	13(52)	33(40.7)		

Thus, several patients with preoperatively blocked ET on Valsalva maneuver had a change in ET function postoperatively. A greater number of patients with blocked ET showed change when assessment was done by Valsalva maneuver. However, the difference between the groups was not found to be statistically significant (p=0.32).

When ET function was assessed by Valsalva maneuver preoperatively and by tympanometry postoperatively, the change in ET function was more apparent (Table 18), particularly for patients who had a blocked ET preoperatively. An ‘A’ tympanogram was interpreted as an intact tympanic membrane with a patent ET.

Table 18: Comparing the change in ET function assessed by Valsalva maneuver preoperatively and tympanometry postoperatively

Preoperative Valsalva maneuver test result	Change present in ET function assessed by tympanometry (%)	No change in ET function assessed by tympanometry (%)	Test used for significance	P value
Patent	9(30.0%)	39(73.5%)	Pearson Chi-square without Yates correction	< 0.001
Not patent	21(70.0%)	14(26.4%)		

When assessed by Valsalva maneuver preoperatively and tympanometry postoperatively, if a patient had a blocked ET preoperatively there was a higher chance of it changing than if a patient had a patent tube ($p<0.001$). Conversely, if a patient had a patent ET preoperatively, there was not much change in the ET function postoperatively. The difference between the two groups those with patent and non-patent ET was statistically significant ($p<0.001$).

The analysis was repeated using the inflation deflation test preoperatively and tympanometry postoperatively (Table 19). Here again, an A tympanogram with an intact tympanic membrane was indicative of a patent ET.

Table 19: Comparing the change in ET function assessed by inflation deflation test preoperatively and tympanometry in the postoperative period

Preoperative inflation deflation test result	Change present in ET function assessed by tympanometry(%)	No change in ET function assessed by tympanometry (%)	Test used for significance	P value
Patent	10 (22.2)	25 (65.8)	Pearson Chi-square without Yates correction	< 0.001
Not patent	35 (77.8)	13(34.2)		

Change in ET function following surgery was also assessed by the inflation deflation test. Here again, patients with preoperatively diagnosed ET block by the inflation deflation test were more likely to show a change in ET function than those with preoperatively patent ET. This difference between those with preoperatively patent and blocked ET was statistically significant ($p< 0.001$).

We then analysed the data to see if the preoperative ET function test result was predictive of closure of the perforation. The analysis was done first with the results of the inflation deflation test and then with the Valsalva maneuver results.

Table 20: Association between preoperative inflation-deflation test result and successful closure of perforation

Preoperative inflation deflation test result	Intact tympanic membrane	Residual perforation	Test used for significance	p-value
Patent	35 (42.2%)	5 (21.7%)	Pearson Chi- square without Yates correction	0.074
Not patent	48 (57.8%)	18 (78.3%)		

Although the patients with non-patent ET had a higher rate of residual perforation (78.3%), the difference between the two groups was not significant (p=0.07).

Table 21: Association between preoperative Valsalva test result and successful closure of perforation

Pre OP Valsalva maneuver test result	Intact tympanic membrane.	Residual perforation	Test used for significance	p-value
Patent	48 (57.8%)	12 (52.2%)	Pearson Chi- square without Yates correction	0.628
Not patent	35 (42.2%)	11 (47.8%)		

The same association was looked at with preoperative Valsalva maneuver and a similar result was seen. So the ET function does not impact on successful closure of perforation. This could be because a number of patients had a change in ET function following closure of the perforation.

DISCUSSION

The etiopathology of middle ear infection can be attributed to many causes of which ET dysfunction is considered to be a very important factor. Middle ear infection leads to further edema and obstruction of the ET.[5] Whether ET dysfunction is a cause or a result of chronic otitis media has been a matter of much debate. Several methods like Valsalva maneuver, Toynbee manoeuvre, ET catheterization, Politzerisation, fluroscein dye method, saccharine test sonotubometry, inflation deflation method, and tympanometry have been described for the assessment of the ET function and different results have been obtained. Among all these tests, the Valsalva test and the inflation deflation test have proved to be the most reliable and popular.[55,56]

ET block is usually transient in many patients with mucosal CSOM. In normals, too, there is diurnal variation in ET patency. Brattmo et al [57] study showed that middle ear pressure regulation is a dynamic process and that at various periods during the day and the night, the ET could be blocked or patent. He also found that there was a significant difference in the diurnal changes in the middle ear pressure in patients with central perforation compared to those with normal healthy ears.

The results of different tests of ET function often show disparate results. This is a big drawback when comparison of results of ET function using different techniques is performed. MacKinnon[58] found that a number of patients who had good ET function on Valsalva maneuver had a poorly functioning ET on the aspiration deflation study. In the present study we found, however, that there was a good correlation between the results of the Valsalva test and inflation deflation test. Postoperatively, however, the results of

tympanometry and Valsalva test were quite disparate. One of the reasons for this could be that the manoeuvre was not performed properly. There could have been a number of patients who despite adequate training could not perform a proper Valsalva and were labelled as having blocked ET. Another reason could be that the ET function is variable and can give different results at different points of time.

The Valsalva maneuver is simple to perform and can be performed at the clinic or bedside and does not require much time to evaluate. Many authors have utilised this technique to evaluate ET function. It is an easily performed test not requiring any extra equipment and hence could be routinely performed for all patients undergoing middle ear surgery. Tos[55] studied both squamous and mucosal chronic otitis media using the Valsalva maneuver and found that the poorest function was in those with squamous disease and active mucosal chronic otitis media. The dysfunction was least in those with inactive chronic mucosal otitis media. Andreasson and Harris[56] in their study felt, however, that training is needed to perform a Valsalva maneuver. The Valsalva maneuver does not reflect on the normal physiological function of the ET. The present study is comparable with results of other studies. When Valsalva maneuver was used to assess the ET patency, many authors found that the ET was patent in the majority [55, 56, 57, 58, 59]. The exact amount of pressure generated while doing Valsalva is not measured. The pressures generated are high and may possibly force open the ET. The major inference that we can make from a positive Valsalva is that there is no organic stenosis of ET.

ET function assessed by aspiration deflation method is considered to be a more physiological test. Choi et al [52] showed that in patients with mucosal chronic otitis media the ET was more likely to be blocked on modified inflation deflation test. He also showed that mucosal swelling and granulations were more frequently found in such patients.

MacKinnon[58] in his study using this test found that with a negative middle ear pressure test, more patients with mucosal chronic otitis media showed a blocked ET.

Virtanen[60] in his study using this test, showed that most of the patients with mucosal chronic otitis media had a blocked ET with the inflation deflation method. In the present study, too, we found that most patients had a blocked ET on the inflation-deflation test. The block was more evident when negative pressure was applied. In contrast, Andreasson and Harris[56] in their study found most patients to have a patent ET on aspiration deflation test. This could be due to the short duration for doing the test, which do not pick up fluctuations in the ET function during a longer period and also other methods like yawning, moving the jaw or autoinflation which equilibrate the underpressure.

Although our null hypothesis for this study was that there was no change in ET function with surgery, it was found on analysis of the results (when preoperative Valsalvamaneuver and inflation deflation were compared with postoperative tympanometry) that many of the non-patent ET became patent following surgery. This was quite different from those patients whose ET was patent preoperatively and this difference was statistically significant ($p<0.001$). This disproved our null hypothesis. However, when the results of preoperative and postoperative Valsalva were compared, the changes were not significant. Similarly, Andreasson and Harris[56] found that there was no change in a patient's ability to perform Valsalva maneuver and hence there was no change in ET function when assessed by Valsalva maneuver. However there was a significant change in ET function when assessed with the aspiration deflation test. Since none of their patients with a preoperative blocked ET had granulations or polyps blocking the ET, they suggested that the change in ET function was due to the change in the

environment in the closed middle ear cavity resulting in a change in the middle ear mucosal lining. Also, a pressure difference occurs across the ET in a closed middle ear cavity which might act as a trigger for the ET to regain function. Our study clearly showed that ET function changed after surgical closure of the tympanic membrane perforation. The change was seen in a far greater number of patients than that showed by other studies.

Tos[55] in his study also showed that ET function changed for a number of patients after surgery. In a long term follow up, patients who persisted to have poor postoperative ET function had a higher chance of developing retractions and adhesions of the tympanic membrane. Choi et al[52] in their study of patients with mucosal CSOM who underwent myringoplasty or tympanoplasty also showed that ET function changed after surgery. It was suggested that removal of mucosal edema and granulations blocking the ET probably resulted in the change in ET function. In contrast, the number of patients with change in ET function in Virtanen's[60] study comparing preoperative aspiration deflation with postoperative tympanometry, were not many. His study does not support the hypothesis that ET function changes after surgery. He suggested that this could not occur without intervention at the tubal orifice during surgery. He also suggested that the small pressure range used in tympanometric studies may be insufficient to move a tympanic membrane and hence may be inaccurate in assessing the normal aeration of middle ear.

The success rate of perforation closure in various studies in which ET function tests have been done varies from 74.5 %to 94.8% [59, 52]. In the present study the rate of tympanic membrane closure was 78.4%. The relatively high rate of residual perforation (21.6%) may be related to the fact that surgery was performed by surgeons with varying degree of skill, possible presence of infection introduced in the perioperative period,

resistant organisms and, of course, poor ET function. The importance of poor ET function as an etiological factor for residual perforation has been stressed by several authors.[50, 54, 59] Some authors have evaluated ET function in relation to successful closure of perforation in mucosal chronic suppurative otitis media also. Depending on the test used to assess ET function, variable results were obtained.

The effect of ET function as assessed by the Valsalva maneuver on successful closure of tympanic membrane perforation was assessed in the present study. There appeared to be no correlation between preoperative Valsalva test result and the presence of either an intact tympanic membrane or residual perforation. Similar results were obtained by other authors.[56] In the study by Reimer et al[59], however, a patent ET on Valsalva was more likely to be associated with an intact tympanic membrane. His study also showed that ears with a combination of positive Valsalva and a functional volume of more than 2 ml had a better healing rate than those with a negative Valsalva and volume of less than 2 ml. There was no association between a blocked ET preoperatively and residual perforation.

When the inflation deflation technique was used, some authors MacKinnon[58] found that healing was much more in patients with a good preoperative ET function than those who had bad ET function. A dry middle ear without any signs of infection and with a good or moderate preoperative ET function had more chance of healing after surgery. However on longer follow up in spite of a good ET function, the authors found that there was a chance of developing recurrent otitis media and tympanic membrane perforation in some individuals (10%). Choi et al[52] showed that a good ET function was related to well aerated middle ear with an 'A' type tympanogram and a good tympanic membrane compliance. Presence of middle ear granulations leads to defective gas exchange and can

also block the ET. Re-perforations in his study were seen in patients with diseased middle ear mucosa. Holmquist's[54] study also showed a good correlation of preoperative ET function with results of myringoplasty. However Andreasson and Harris[56] showed that there was no correlation between healing and preoperative ET function when assessed with the aspiration deflation method. According to the authors, the available ET function tests are of no value for selecting patients suitable for middle ear surgery. In the present study, we found an increased percentage of residual perforation in those with blocked ET when assessed by the inflation deflation test. However, this did not reach statistical significance (p=0.07)

CONCLUSION

In conclusion, the results of our study on preoperative ET function in patients with mucosal CSOM show that the results of evaluation of ET function vary with the technique used. There appears to be no single test of ET function which may be consistently relied upon. However, the Valsalva tests and inflation deflation test are the easily performed and most popular.

We arrived at 3 important conclusions in this study.

Firstly, the ET appeared to be blocked in most (62.6%) patients with mucosal CSOM when assessed preoperatively with the inflation deflation test, whereas most appeared tube patent (54.9%) with the Valsalva maneuver.

Secondly, there was a change in the ET function after surgery which was significant ($p<0.001$) when assessed with both Valsalva and inflation deflation preoperatively and tympanometry post operatively. The function of the ET tends to change from blocked towards patency, possibly due to the removal of the disease and return of normal physiological state after surgery.

Finally, there was an increased tendency of a patient with blocked ET to develop a residual perforation, although this did not reach statistical significance.

BIBLIOGRAPHY

1. Bluestone CD. Epidemiology and pathogenesis of chronic suppurative otitis media: implications for prevention and treatment. Int. J. Pediatr. Otorhinolaryngol. 1998;42:207–23.

2. Holmquist J. Middle ear ventilation in chronic otitis media. Arch Otolaryngol. 1970;92:617–23.

3. Vartiainen E, Nuutinen J. Success and pitfalls in myringoplasty: follow-up study of 404 cases. Am J Otol. 1993 ;14:301–5.

4. Jonathan D. The predictive value of eustachian tube function (measured with sonotubometry) in the successful outcome of myringoplasty. Clin Otolaryngol Allied Sci. 1990 ;15:431–4.

5. Bluestone CD. Historical Perspective. In: Larissa Byj. Editor. Eustachian Tube Structure, Function, Role in Otitis Media. 2nd ed. Vol 2. Hamilton. London BC Decker Inc. 2005.p 4-9.

6. Eustachius B. Epistola de auditusorganis. Arch Otolaryngol 1944. (40):123.

7. Toynbee J. On Disease of the Internal Ear.Lond J Med. 1852 ;4:724–730.

8. Politzer A. Methods of Examination of Middle ear. In: Sir William Dalby. Editor.Politzer's Text Book of the Diseases of the Ear and adjascent organs. 3rd ed. Philadelphia and New york. Lea Brothers & Co.1894. p. 113-124

9. Swarts JD, Rood SR, Doyle WJ. Fetal development of the auditory tube and paratubal musculature. Cleft Palate J. 1986 ;23:289–311.

10. Bluestone CD. Anatomy and Physiology of the Eustachian Tube. In: Cummings Otolaryngology Head and Neck Surgery. 3rd ed. Vol 4.p 3003-3012

11. Sadler-Kimes D, Siegel MI, Todhunter JS. Age-related morphologic differences in the components of the eustachian tube/middle ear system. Ann. Otol. Rhinol. Laryngol.1989 ;98:854–858.

12. Proctor B. Embryology and anatomy of the eustachian tube. Arch Otolaryngol.

1967;86:503–514.

13. Macbeth R. Some thoughts on the eustachian tube. Proc. R. Soc. Med. 1960 ;53:151–61.

14. Hasebe S, Sando I, Orita Y. Proximity of carotid canal wall to tympanic membrane: a human temporal bone study. Laryngoscope.2003 ;113:802–7.

15. Prades JM, Dumollard JM, Calloc'h F, Merzougui N, Veyret C, Martin C. Descriptive anatomy of the human auditory tube. SurgRadiol Anat. 1998;20:335–40.

16. Luntz M, Pitashny R, SadJ. Cartilage in the bony portion of the eustachiantube.Eur Arch Otorhinolaryngol.1990 ;247:56-59.

17. Sudo M, Sando I, Ikui A, Suzuki C. Narrowest (isthmus) portion of eustachian tube: a computer-aided three-dimensional reconstruction and measurement study. Ann. Otol. Rhinol. Laryngol.1997 ;106:583–8.

18. Lim DJ. Functional morphology of the mucosa of the middle ear and Eustachian tube. Ann. Otol. Rhinol. Laryngol.1976 ;85:36–43.

19. Tomoda K, Morii S, Yamashita T, Kumazawa T. Deviation with increasing age in histologic appearance of submucosal glands in human eustachian tubes. ActaOtolaryngol.1981 ;92:463–7.

20. Swarts JD, Rood SR. The morphometry and three-dimensional structure of the adult eustachian tube: implications for function. Cleft Palate J. 1990;27:374–81.

21. Rood SR, Doyle WJ. Morphology of tensor velipalatini, tensor tympani, and dilatator tubae muscles. Ann. Otol. Rhinol. Laryngol. 1978;87:202–10.

22. Lupin AJ. The relationship of the tensor tympani and tensor palati muscles. Ann. Otol. Rhinol. Laryngol.1969 ;78:792–6.

23. Sudo M, Sando I, Suzuki C. Three-dimensional reconstruction and measurement study of human eustachian tube structures: a hypothesis of eustachian tube function. Ann. Otol. Rhinol. Laryngol.1998 ;107:547–54.

24. Cunsolo E, Marchioni D, Leo G, Incorvaia C, Presutti L. Functional anatomy of the Eustachian tube. Int J Immunopathol Pharmacol. 2010 ;23:4–7.

25. Ostfeld EJ, Silberberg A. Transient pressure changes in the middle ear. Arch. Otolaryngol. Head Neck Surg. 1991 ;117:1390–4.

26. Elner A, Ingelstedt S, Ivarsson A. The normal function of the eustachiantube.A study of 102 cases.ActaOtolaryngol.1971 ;72:320–8.

27. Bluestone CD, Beery QC. Concepts on the pathogenesis of middle ear effusions. Ann. Otol. Rhinol. Laryngol.1976 ;85:182–6.

28. Sadé J, Eliezer N. Secretory otitis media and the nature of the mucociliary system. ActaOtolaryngol.1970 ;70:351–7.

29. Hentzer E. Ultrastructure of the middle-ear mucosa in chronic suppurative otitis media. J Laryngol Otol. 1972;86:447–56.

30. Brookler KH, Birken EA. Surface tension lowering substance of the eustachian tube. Laryngoscope.1971 ;81:1671–3.

31. Pau HW, Sievert U, Just T, Sadé J. Pressure changes in the human middle ear without opening the eustachian tube. Acta Otolaryngol.2009 ;129:1182–6.

32. Bluestone CD. Pathogenesis of otitis media: role of eustachian tube. Pediatr. Infect. Dis. J. 1996 ;15:281–91.

33. Alper CM, Kitsko DJ, Swarts JD, Martin B, Yuksel S, Cullen Doyle BM, et al. Role of the mastoid in middle ear pressure regulation. Laryngoscope.2011 ;121:404–8.

34. Buchman CA, Doyle WJ, Pilcher O, Gentile DA, Skoner DP. Nasal and otologic effects of experimental respiratory syncytial virus infection in adults.Am J Otolaryngol.2002 ;23:70–5.

35. Swarts JD, Alper CM, Seroky JT, Chan KH, Doyle WJ. In vivo observation with magnetic resonance imaging of middle ear effusion in response to experimental underpressures. Ann. Otol. Rhinol. Laryngol.1995 ;104:522–8.

36. Boswell JB, Nienhuys TG. Patterns of persistent otitis media in the first year of life in aboriginal and non-aboriginal infants. Ann. Otol. Rhinol. Laryngol.1996 ;105:893–900.

37. Prevention of hearing impairment from chronic otitis media,Report of a WHO/CIBA Foundation Workshop, London: 19-21 November 1996.

38. Sano S, Kamide Y, Schachern PA, Paparella MM. Micropathologic changes of pars tensa in children with otitis media with effusion. Arch. Otolaryngol. Head Neck Surg. 1994 ;120:815–9.

39. Nadol, Merchant SN, M.D Joseph BN Jr. Infections. In: Joseph BN Jr. Editor. Schucknect's Pathology of the Ear.3rd ed. part-2.PMPH-USA. 2010. p. 292 -96.

40. Vartiainen E. The results of chronic ear surgery in a training programme. ClinOtolaryngol Allied Sci. 1998 ;23:177–80.

41. McGrew BM, Jackson CG, Glasscock ME 3rd. Impact of mastoidectomy on simple tympanic membrane perforation repair. Laryngoscope.2004 ;114:506–11.

42. Mishiro Y, Sakagami M, Takahashi Y, Kitahara T, Kajikawa H, Kubo T. Tympanoplasty with and without mastoidectomy for non-cholesteatomatous chronic otitis media. Eur Arch Otorhinolaryngol.2001 ;258:13–5.

43. Bluestone CD, Shurin PA. Middle ear disease in children.Pathogenesis, diagnosis, and management.Pediatr.Clin. North Am. 1974 ;21:379–400.

44. Linstrom CJ, Silverman CA, Rosen A, Meiteles LZ. Eustachian tube endoscopy in patients with chronic ear disease.Laryngoscope.2000 ;110:1884–9.

45. Jerger J. Clinical experience with impedance audiometry.Arch Otolaryngol.1970 ;92:311–24.

46. Rogers RL, Kirchner FR, Proud GO. The evaluation of Eustachian tubal function by fluorescent dye studies.Laryngoscope.1962 ;72:456–67.

47. el-Guindy A. Manometric and endoscopic study of tubal function in drum perforation. Am J Otol. 1993 ;14:580–4.

48. van der Avoort SJC, van Heerbeek N, Zielhuis GA, Cremers CWRJ. Sonotubometry: eustachian tube ventilatory function test: a state-of-the-art review. Otol. Neurotol. 2005 ;26:538–43;

49. Flisberg K. Controlled Ear Aspiration of Air - A “physiological test of the tubal funtion.”Acta otolaryng.Suppl. 182.

50. Takahashi H, Sato H, Nakamura H, Naito Y, Umeki H. Correlation between middle-ear pressure-regulation functions and outcome of type-I tympanoplasty. Auris Nasus Larynx.2007 ;34:173–6.

51. Uzun C, Cayé-Thomasen P, Andersen J, Tos M. Eustachian tube patency and function in tympanoplasty with cartilage palisades or fascia after cholesteatoma surgery. Otol. Neurotol. 2004 ;25:864–72.

52. Choi SH, Han JH, Chung JW. Pre-operative Evaluation of Eustachian Tube Function Using a Modified Pressure Equilibration Test is Predictive of Good Postoperative Hearing and Middle Ear Aeration in Type 1 Tympanoplasty Patients. Clin Exp Otorhinolaryngol.2009 ;2:61–5.

53. Kurien R, Chrisolyte S, RupaV.Inflation-deflation test as a preditor of aditus patency in patients with chronic suppurative otitis media. Indian J Otolaryngol Head Neck Surg. 200961:169–72.

54. Holmquist J. Auditory tubal function in chronic middle ear disease and the role of tubal function in myringoplasty. In: International audiology Vol 9;1970; 197-201

55. Tos M. Impartance of eustachian tube function in middle ear surgery. Ear nose and throat journal 1998;77:744-747

56.Adreasson L. and Harris S. Middle ear mechanics and eustachian tube function in tympanoplasty. Acta Otolaryngol, Suppl.1979; 360: 141-147

57. Brattmo M, Tideholm B, Carlborg B. Chronic tympanic membrane perforation: Middle ear pressure and tubal function. Acta Otolaryngol 2003; 123: 569-574

58. MacKinnon D M. Relationship of preoperative eustachian tube function to myringoplasty. Acta Otolaryngol 1970; 69: 100-106

59. Reimer A, Andreasson L, Harris S, Ivarsson A, Tjernstrom O. Tubal function and surgery in chronic otitis media. A study on predictive value of testing tubal function, Valsalva maneuver and volume of ear spaces. Acta Otolarngol 1988; 449: 127-130

60. Virtanen H, Palva T, Jauhiainen T. The prognostic value of Eustachian tube measurements in Tympanoplasty surgery. Acta Otolaryngol 1980; 90: 317-323

APPENDIX A

Proforma for study on Eustachian tube function before and after surgery for mucosal chronic suppurative ottitis media

Name: Age / Sex:

Hospital number:

Side: Unilateral / Bilateral Right Left

Address:

Phone No:

Symptoms: Right Left

Duration of ear discharge: months years

Last episode of discharge:

Associated ear symptoms: Tinnitus Vertigo Earache Hearing loss

Nose and throat complaints:

Other Co morbidities:

Previous surgeries: Right Left

Signs:

Perforation size: Pinpoint Small Large Subtotal

Middle ear mucosa: Dry, Moist, Discharging, Edematous Polypoidal Granulations

Pre OP Audiogram:

	500Hz **R L**	**1Khz**	**2Khz**	**3Khz**	**PTA average**	**Air bone gap average**
AC						
BC						

Pre OP X-ray mastoids: Sclerosis Cellular

Pre OP Valsalva maneuver: ET patent, ET blocked

Pre OP Inflation Deflation test:

Residual pressure	**Present** **R** **L**	**Absent** **R** **L**
Inflation		
Deflation		

Surgery done:

Date of surgery:

Surgical findings:

Middle ear Granulations Edematous mucosa Tympanosclerosis

Glue congested

Mastoid Granulations Edematous mucosa Tympanosclerosis

Contracted sclerotic cellular

Aditus patency: Patent Block

Ossicular erosion: Malleus: Handle of Malleus; Head of Malleus, Medialised

Incus: Lenticular process; Long process; Body; Short process

Stapes: Head; Crura

Cartilage used: Pallisades Single piece

Postop:

Number of months postop:

Otomicroscopy findings:

Graft: Intact Residual perforation Retracted Tympanosclerosis

Post OP Audiogram:

	500Hz **R L**	**1Khz**	**2Khz**	**3Khz**	**PTA average**	**Air-bone gap average**
AC						
BC						

Post OP Tympanometry: A As Ad B C

Post OP Valsalva: ET patent ET blocked

APPENDIX B

PATIENT INFORMATION SHEET

You are being requested to participate in a study to assess your Eustachian tube (ET) function before and after your ear surgery. ET connects the ear to the nose and helps to equalize the pressure in the ear. Some studies have shown that ET function may have a bearing on the outcome of surgery (hearing, graft uptake etc.) Therefore we are conducting this study to see if this is true.

If you take part what will you have to do?

You will be undergoing a surgery for the closure of your ear drum perforation with or without operation of the bone. If you agree to participate in this study we will be conducting the following test before surgery:

1. Inflation-deflation test. This test will show the ET function in an ear with a perforation.
2. Valsalva maneuver. This is another test of eustachian tube function.

After surgery the following tests will be done at 3 months when you return for your regular postoperative check up

1. Valsalva maneuver
2. Tympanometry. This test will show the ET function in an ear without a perforation.

The tests are non-invasive and not painful

Can you withdraw from this study after it starts?

Your participation in this study is entirely voluntary and you are also free to decide to withdraw permission to participate in this study. If you do so, this will not affect your usual treatment at this hospital in any way.

What will happen if you develop any study related injury?

The tests done in this study are diagnostic and do not have any risk of injury.

Will you have to pay for the tests?

You will not have to pay for the additional tests

Will your personal details be kept confidential?

The results of this study will be published in a medical journal but you will not be identified by name in any publication or presentation of results. However, your medical notes may be reviewed by people associated with the study, without your additional permission, should you decide to participate in this study.

If you have any further questions, please ask Dr. Lisa Mary Cherian, (tel: 04162283483 mob: 09486233616) or email: lisamarycherian@gmail.com

INFORMED CONSENT TO TAKE PART IN A CLINICAL STUDY

Study Title: Effect of ET function in postoperative graft uptake and hearing improvement

Study Number:

Participant's name:

Date of Birth / Age (in years):

I_____

_____, son/daughter of _____

(Please tick boxes)

declare that I have read the information sheet provide to me regarding this study and have clarified any doubts that I had. []

I also understand that my participation in this study is entirely voluntary and that I am free to withdraw permission to continue to participate at any time without affecting my usual treatment or my legal rights [ ]

I also understand that I will have to undergo some additional tests as part of the study for which I will not be charged. I also understand that I will receive free treatment for any

study related injury or adverse event but I will not receive any other financial compensation [ ]

I understand that the study staff and institutional ethics committee members will not need my permission to look at my health records even if I withdraw from the trial. I agree to this access [ ]

I understand that my identity will not be revealed in any information released to third parties or published [ ]

I voluntarily agree to take part in this study [ ]

Name:

Signature:

Date:

Name of witness:

Relation to participant:

Date:

DATA SHEET OF STUDY PATIENTS (n=182)

S.No.	NAME	AGE	Sex M-1, F-2	Affected side U-1, B-2	Ear discharge Y-1, N-2	Duration (yr)	Last episode of discharge (days)	Tinnitus Y-1, N-2	Nose Nil -1, Allergy-2	Comorbidities Y-1, N-2	Previous surgery Y-1, N-2	Perforation size S/M-1, L/ST-2	Middle ear mucosa D-1, M-2
1	PARAMESHWARI	43	2	1	1	3	15	2	1	2	2	2	2
2	DEEPA M -1	32	2	2	1	5	30	2	1	2	2	2	2
3	BHARATHIRAJA 1 * L	31	1	2	1	0.5	90	1	1	2	2	2	1
4	JAYANTHA MONDAL	28	1	1	1	8	2190	1	1	2	2	1	1
5	MANIVEL M	21	1	2	1	6	120	1	1	2	2	2	1
6	SHAUKAT ALI -R	44	1	2	1	35	1	2	1	2	2	2	1
7	BIDAN DAS	44	1	2	1	30	3	2	1	2	2	2	2
8	VIJAYA KUMARI	30	2	2	1	20	30	2	1	1	2	2	2
9	BISHNU CHARAN KARMAKAR	33	2	2	1	18	2555	2	1	2	2	2	1
10	BAIKASHI GIRI	20	2	2	1	15	1	2	1	2	2	2	2
11	RINA BASAK	27	2	1	1	7	4	1	1	2	2	2	1
12	SATHYA SUMITHA	28	2	1	1	0.4	1	2	1	2	2	2	1
13	DIPANKAR DAS 1	39	1	1	1	25	7	2	1	2	1	2	1
14	JAYACHITRA -1	20	2	2	1	15	2	2	2	2	2	2	2
15	KABITHA MONDAL	27	2	1	1	7	1	2	1	2	2	2	2
16	MD.TAZOL ISLAM	29	1	1	1	20	30	1	1	2	2	2	2
17	MANJUNATH	22	1	2	1	10	120	1	1	2	2	2	1
18	KUMAR -R	22	1	2	1	25	60	2	1	2	2	1	1
19	KUMAR -L	31	1	2	1	25	60	2	1	2	2	1	1
20	SUKUMAR DEY PODDER	50	1	1	1	1	90	1	1	2	2	2	1
21	MINATHI SARKAR	26	2	1	1	5	10	2	2	2	2	2	2
22	KOLANJI	49	2	1	1	5	1	1	2	1	2	2	2
23	AZIZA AKHTAR	26	2	2	1	24	1	1	1	2	2	2	2
24	SHANTHI DEVI	55	2	1	1	2	5	2	2	1	2	1	1
25	SAPAN DEY	33	1	1	1	23	2	1	2	2	2	1	2
26	ABDUL RAHMAN	23	1	1	1	17	210	2	1	2	2	2	1
27	MOUNI S	17	1	1	1	4	30	2	1	2	1	1	2
28	JAHANGIR JAMADAR	34	1	2	1	30	20	1	1	2	2	2	2
29	DIPAK GUPTA	46	1	1	1	0.75	30	1	2	2	2	2	1
30	BHUVANESHWARI	36	2	1	1	32	10	2	1	1	2	2	2
31	SATHYA	23	2	1	1	5	2	1	2	2	2	1	2
32	SOUMEN GARANG	27	1	1	1	3	90	1	1	2	2	1	1
33	NAZRUL ISLAM	18	1	2	1	10	60	2	1	2	2	2	2
34	KALAVATHI	33	2	2	1	10	30	1	1	2	2	1	1
35	RABIRAM TIMU	37	1	1	1	10	60	1	2	2	1	1	1
36	PUSHPARANI-1	48	2	1	1	2	15	2	1	1	2	2	1
37	TATAPUDI PRAKASH	29	1	1	2	N	N	2	1	2	2	2	1
38	KISHORE KUMAR GHOSH	19	1	2	1	6	730	1	1	2	2	1	1
39	SHAYAM LAL AGARWAL	28	1	1	1	15	30	2	2	2	2	1	2
40	RAJESHWARI *	31	2	1	1	1	210	1	2	2	2	2	1

S.No.	Avg AB gap R & L	SNHL Y-1, N-2	PreOPValsalva P-1, NP-2	Pre OP I&D (P,P-1), (P,NP/NP,NP/NP,P-2)	Tympanic remanent (Tsp/ode)-1,N-2	Mastoid C-1, S-2	Antrum N-1, OE-2	Aditus P-1, B-2	Aditus made P-1,PP-2,B-3	Ossicles N-1, (ER,TS-2),NA-3	N-1,(R/A-2),NA-3	Catlilage/bone Y-1,N-2	Follow up (mnths)	Post OP graft (N,&GRN-1), CP-3	AB gapR&L	Tymps (A-1),(C-2),(B-3),(CP-4)	POST Valsalva P-1,Np-2
1	23.75	1	2	2	2					1	1	2	3	1	13.75	1	2
2	38.75	2	2	2	2	2	2	2	1	1	1	2	3	3		4	2
3	18.75	2	1	1	2					1	1	2	14	1	26.25	1	1
4	23.75	2	1	1	2					1	1	2	12	1	0	1	1
5	25	1	2	2	1	2	2	2	1	1	1	2	7	1	25.25	3	2
6	41.7	1	2	2	2	2	1	2	2	1	1	2	7	1	28.4	2	2
7	11.7	1	2	2	2					1	1	2					
8	47.5	2	1	1	2	2	2	2	1	2	1	2	4	1	2.5	1	1
9	35	2	1	2	2	2	1	1	1	1	1	2					
10	25	2	2	1	2	2	1	1	1	1	1	2					
11	20	2	2	1	1					1	1	2					
12	16.7	2	1	1	2	1	1	1	1	1	1	2	6	1	0	1	1
13	23.75	2	2	2	2	2	1	1	1	2	2	2	12	1	21.25	3	2
14	22.5	2	1	2	2	1	1	1	1	1	1	2	3	3		4	1
15	31.6	2	1	1	2	2	2	2	2	1	1	2					
16	28.3	1	2	1	2					2	2	2					
17	0	2	2	2	2					3	3	2	3	1	0	1	2
18	38.3	1	1	2	2					3	3	2					
19	45	1	2	2	2					3	3	2					
20	12.5	1	2	2	2					1	1	2	8	1	6.25	1	2
21	18.3	2	2	2	2					1	1	2	6	1	0	1	1
22	24.9	2	1	1	1	2	1	2	2	3	3	2	3	1	0	2	2
23	50	2	2	2	2	2	2	2	1	2	2	1					
24	35	2	2	1	2					1	1	2	6	1	25	1	1
25	21.6	2	2	2	2	2	1	1	1	1	1	2					
26	26.6	2	2	1	1					1	1	2	4.5	1	18.3	3	2
27	26.6	2	1	2	1	2	1	1	1	1	1	2	4.5	1	0	1	1
28	50	2	2	2	2	2	2	2	2	1	1	2	4.5	3		4	2
29	15	2	1	1	2					1	1	2	6	1	0	1	1
30	33.3	1	2	1	2	2	2	2	3	2	2	2					
31	25	1	2	1	1	1	1	2	1	1	1	2	3.5	1	11.7	2	1
32	23.3	2	1	2	2	1	1	1	1	1	1	2	3	1	0	1	1
33	52.5	2	2	2	2	2	2	2	3	1	1	2	12	3		4	2
34	20	2	1	1	2					1	1	2	5	1	0	1	1
35	20	2	1	1	2					1	1	2					
36	26.25	2	1	1	2	1	1	1	1	1	1	2	11	1	0	1	1
37	28.3	2	2	2	1					1	1	2					
38	23.3	2	1	1	2					1	1	2	4	1	0	1	1
39	26.7	2	1	2	2					1	1	2	3	1	0	1	1
40	23.3	2	2	2	1					1	1	2	4	1	0	1	2

S.No.	NAME	AGE	Sex M-1, F-2	Affected side U-1, B-2	Ear discharge Y-1, N-2	Duration(yr)	Last episode of discharge (days)	TinnitusY-1, N-2	NoseNil -1, Allergy-2	Comorbidities Y-1, N-2	Previous surgeryY-1, N-2	Perforation size S/M-1, L/ST-2	Middle ear mucosa D-1, M-2
41	REBA BAR	42	2	1	1	1	120	1	1	2	2	2	1
42	SYED BASHA	44	1	1	1	6	4	1	1	2	2	2	2
43	KANCHAN ROY	44	1	2	1	1.5	547	2	1	2	2	2	1
44	GOPAL RAO	26	1	1	1	10	730	2	2	2	1	1	1
45	ARUP GOURAI *	22	1	1	1	10	30	2	1	2	2	1	1
46	SANTHOSH GOPE	24	1	2	1	18	365	2	1	2	2	2	2
47	BABITA CHULYA	38	2	1	1	25	365	2	1	1	1	1	1
48	GITA RANI MONDAL	55	2	2	1	30	1	1	2	1	2	2	2
49	SELVAM C	30	1	2	1	2	30	2	1	2	2	2	1
50	SUMAN KUMARI	25	2	2	1	4	30	2	2	2	2	2	2
51	SIVALINGAM	18	1	1	1	15	30	2	2	2	2	2	2
52	SYFULLA	30	1	1	1	10	90	2	1	2	2	2	2
53	TANUSHREE MONDAL	27	2	1	1	3	60	2	2	1	2	2	1
54	TANUMOUY MAITY	20	1	1	1	3	60	2	1	2	2	1	2
55	BERNAULI MAHATO ***	27	2	1	1	20	1	2	1	2	2	2	2
56	V GOVINDASWAMY	57	1	1	1	5	90	2	2	2	2	1	1
57	MALA MONDAL	42	2	2	1	26	120	1	1	2	2	1	1
58	RUMKI NANDY	26	2	1	1	15	30	1	2	2	2	1	2
59	ALTAB MONDAL	33	1	1	1	3	60	1	1	2	2	2	1
60	PRANABESH PRADAN	36	1	1	1	28	120	2	2	2	2	1	1
61	KALAVATHY *	38	2	1	2	N	N	2	1	2	2	2	1
62	MALLIGA .M	40	2	2	1	30	2	2	1	2	2	2	1
63	LAKSHMI	36	2	2	1	2	4	2	1	2	2	2	2
64	NILAVATI A	36	2	2	1	6	30	1	1	2	2	2	1
65	SIMANT KUMAR	22	1	1	1	15	60	2	2	2	2	2	2
66	ELUMALAI - I	36	1	2	1	20	21	1	1	2	2	2	2
67	PHANI KALITA	46	1	2	1	30	90	2	1	1	1	2	2
68	YUVARAJ - LEFT	23	1	2	1	20	30	1	2	2	2	2	1
69	ASHIS KUMAR ADAK	35	1	1	1	20	1	2	2	1	2	2	2
70	JEEVANANDAM	19	1	1	1	12	90	1	2	2	2	1	1
71	VIGNESH *	19	1	2	1	10	45	2	2	2	2	2	2
72	SHJEO ALAM	39	2	2	1	10	30	1	2	2	2	2	2
73	KHUBLAL MONDAL - 2	40	1	2	1	6	60	1	2	2	2	2	1
74	DHANALAKSHMI	19	2	1	1	7	30	2	1	2	2	1	1
75	DEVIKA M	36	2	1	1	6	30	2	1	2	2	2	2
76	BINA DEVI	54	2	1	1	30	4	2	2	2	2	2	2
77	RAHIMA KHANAM MAZUMDER	43	2	1	1	2	1	2	1	2	2	2	2
78	MARUFA BEGAM	34	2	2	1	20	1	1	2	2	2	2	2
79	WAJIDA	32	2	1	1	5	60	2	2	2	2	2	2
80	RAGHUNATH KUNDU	49	1	2	1	25	20	1	2	2	2	2	2

S.No.	Avg AB gap R & L	SNHL Y-1, N-2	PreOPValsalva P-1, NP-2	Pre OP I&D (P,P-1), (P,NP/NP,NP/NP,P-2)	Tympanic remanent (Tsp/ode)-1,N-2	Mastoid C-1, S-2	AntrumN-1, OE-2	AditusP-1, B-2	Aditus made P-1,PP-2,B-3	OssiclesN-1, (ER,TS-2),NA-3	N-1,(R/A-2),NA-3	Catilage/boneY-1,N-2	Follow up(mnths)	Post OP graft (N,&GRN-1), CP-3	AB gapR&L	Tymps (A-1),(C-2),(B-3),(CP-4)	POST Valsalva P-1,Np-2
41	18.4	1	2	2	2					3	3	2					
42	30	2	2	2	2	2	2	2	3	1	1	2					
43	18.3	2	1	2	2					1	1	2	3	3		4	1
44	40	2	1	1	2					1	1	2	3	1	45	1	1
45	31.6	2	1	2	2					1	1	2	3	1	15	1	1
46	31.6	2	1	2	2					1	1	2	5	1	15	1	1
47	38.4	2	2	1	2					2	1	1	6	1	45	3	1
48	21.6	2	2	2	2	2	1	1	1	1	1	2					
49	33.75	2	2	2	2	2	1	1	1	1	1	2					
50	20	1	2	2	2	1	1	2	1	1	1	2					
51	21.7	2	1	2	2	2	1	1	1	2	1	2	5	1	23.75	1	2
52	60	2	2	2	2	2	1	1	1	2	2	1	3	1	23.4	1	1
53	13.3	2	1	1	2	1	1	2	3	1	1	2	4	1	21.5	2	1
54	15	2	2	1	1					2	2	2	3	1	0	1	2
55	40	1	2	2	2	2	1	1	1	2	2	2	7	1	31.25	2	2
56	23.4	1	1	2	2	2	1	1	1	2	1	2	7	1	8.75	1	1
57	40	2	1	2	2	2	1	1	1	2	1	1	4	3		4	1
58	41.6	1	1	1	2					1	1	2					
59	10	2	1	1	2					1	1	2					
60	15	2	1	1	2	2	1	1	1	1	1	2	4	1	0	1	1
61	13.3	2	1	1	2					1	1	2	4	1	18.3	1	1
62	18.6	2	1	2	2	1	1	1	1	1	1	2	3	1	0	1	2
63	30	1	2	2	2	2	1	2	2	2	1	2	3	1	53.3	3	2
64	20	1	2	2	2					1	1	2	5	1	2.5	1	2
65	31.6	2	2	1	2	2	1	2	2	2	1	2	2	1	0	2	2
66	27.25	2	2	2	1	2	2	2	1	1	1	2	6	1	13.75	1	2
67	45	2	1	1	2	2	2	2	3	1	1	2	4	1	20	3	2
68	33.3	2	1	1	2	2	2	2	3	1	1	2	3	1	0	1	1
69	41.7	1	1	1	2	2	2	2	2	2	2	2	8	1	35	1	1
70	20	2	2	2	2					3	3	2	3	1	0	1	1
71	51.7	2	1	1	2	2	2	2	3	1	1	2	3	1	0	1	1
72	37.5	2	2	2	2	2	2	2	1	1	1	2	4	1	16.25	2	2
73	40	1	2	2	2					1	1	2					
74	33.3	2	1	2	2	2	2	1	1	1	1	2	4	1	18.75	1	1
75	25	2	1	1	2	1	1	1	1	1	1	2	4	3		4	2
76	40	1	1	2	2	2	2	2	3	3	3	2					
77	25	2	2	1	2	2	1	1	1	1	1	2	4	1	13.75	1	2
78	38.4	1	1	1	1	2	2	2	2	2	2	2					
79	33.4	1	2	2	2	2	2	2	1	1	1	2	3	1	10	1	2
80	50	2	1	1	2	2	2	2	1	1	1	2					

S.No.	NAME	AGE	Sex M-1, F-2	Affected sideU-1, B-2	Ear dischargeY-1, N-2	Duration(yr)	Last episode of discharge (days)	TinnitusY-1, N-2	NoseNil -1, Allergy-2	Comorbidities Y-1, N-2	Previous surgeryY-1, N-2	Perforation sizeS/M-1, L/ST-2	Middle ear mucosa D-1, M-2
81	AHMAD DIDAD	21	1	2	1	10	90	2	1	2	2	2	1
82	BHAKTHI PADA GHOSH	49	1	2	1	35	60	2	1	2	1	2	1
83	SURYA PRAKASH GOSH	40	1	1	1	2	30	2	1	2	2	2	2
84	RINA DAS	20	2	2	1	10	730	1	1	2	2	2	2
85	MOHAN V	23	1	1	1	16	365	2	2	2	2	1	2
86	AMAR BARI RIGHT	22	1	2	1	15	30	1	1	2	2	2	2
87	SARASWATHI MONDAL	24	2	2	1	7	90	1	2	2	2	2	1
88	ARUN KUMAR	19	1	2	1	7	270	2	1	2	1	2	2
89	BAKIYARAJ	22	1	1	1	15	60	1	2	2	2	1	1
90	BHAGIRATH GAURAI	40	1	2	1	30	30	1	1	2	2	2	1
91	VENKATESULU	40	1	2	1	20	2555	1	2	1	2	2	1
92	ALEX PANDIAN	16	1	2	1	10	1	2	1	2	2	2	2
93	DEVAN	19	1	2	1	12	30	1	1	2	2	2	1
94	PRIYA	27	2	1	1	1	20	2	1	2	2	2	2
95	BHAKTA DAS	30	2	2	1	20	120	2	1	2	2	2	2
96	RENUKA BISWAS	38	2	1	1	2	21	1	1	2	2	2	1
97	NANDHINI -R	19	2	2	1	15	7	2	1	2	2	2	2
98	NANDHINI - L	19	2	2	1	15	7	2	1	2	2	2	2
99	KALPANA GORAI	40	2	1	1	12	120	2	1	1	2	2	1
100	JEEVITHA	24	2	2	1	15	4	2	1	2	2	2	2
101	PRASANJIT MUKHERJEE	16	1	2	1	14	7	2	1	2	2	2	2
102	NAVLESH KUMAR	19	1	1	1	12	90	1	1	2	2	2	2
103	TAHIRA	50	2	1	1	2	4	1	1	1	2	1	1
104	HARENDRA HAZARIKA	53	1	2	1	3	10	1	1	2	2	2	2
105	POORNIMA UPADHYA	41	2	2	1	3	2	1	2	1	2	2	2
106	ABHAY KUMAR SINGH	34	1	2	1	30	2	2	1	1	2	2	2
107	SWAPAN DATTA	41	1	2	1	10	30	2	1	2	2	2	2
108	FATHICK CHANDRA PRAMANIK L	41	1	2	1	35	60	2	1	2	2	2	2
109	MUNIRATHINAM D	54	1	1	1	1	60	2	1	1	2	2	1
110	NAJARAJ J	33	1	1	2	N	N	2	1	2	2	1	1
111	MUKHESH KUMAR	26	1	1	1	20	120	1	1	2	2	2	1
112	SURESH -1	24	1	2	1	20	120	1	1	2	2	2	2
113	KUMAR SADANAND	23	1	1	1	3	10	2	1	2	2	2	2
114	SAHIDUL ISLAM	34	1	1	1	2	30	2	1	2	1	2	2
115	NIRMALA	32	2	1	1	3	3	2	2	1	2	2	1
116	KUNAL KANTHI	25	1	1	1	2	6	1	1	2	2	2	1
117	FATHIMA	19	2	1	1	15	30	1	1	2	2	2	1
118	SIMA RANI GHORAI	22	2	1	1	5	30	1	1	2	2	2	2
119	SHAUKAT ALI -L	44	1	2	1	3	30	2	1	1	2	2	1
120	RANG KYNSAIRYNJAI	33	1	1	1	6	2	2	1	2	2	2	2

S.No.	Avg AB gap R & L	SNHL Y-1, N-2	PreOPValsalvaP-1, NP-2	Pre OP I&D (P,P-1), (P,NP/NP,NP/NP,P-2)	Tympanic remanent (Tsp/ode)-1,N-2	Mastoid C-1, S-2	AntrumN-1, OE-2	AditusP-1, B-2	Aditus made P-1,PP-2,B-3	Ossicles N-1, (ER,TS-2),NA-3	N-1,(R/A-2),NA-3	Catlilage/boneY-1,N-2	Follow up(mnths)	Post OP graft (N,&GRN-1), CP-3	AB gapR&L	Tymps (A-1),(C-2),(B-3),(CP-4)	POST Valsalva P-1,Np-2
81	32.5	1	2	2	2					1	1	2	3	1	20	3	2
82	45	2	2	2	2	2	2	1	1	2	1	2	7	3		4	1
83	48.75	2	1	2	2					1	1	2	3	3		4	1
84	40	2	1	2	2	2	2	2	2	1	1	2					
85	33.3	2	1	2	2	2	2	2	1	1	1	2	3	3		4	1
86	27.5	2	2	2	2	2	2	2	2	1	1	2	6	3		4	2
87	40	2	1	2	2	2	1	1	1	1	1	2					
88	47.5	2	2	2	2					3	3	2	9	1	17.5	1	1
89	15	2	1	2	1					1	1	2	3	1	0	1	2
90	25	2	1	2	2	2	1	1	1	1	1	2					
91	25	2	2	2	2					1	1	2	9	3		4	2
92	35	2	1	2	2	2	2	1	1	1	1	2	3	1	0	1	1
93	41.6	2	1	1	2	2	2	1	1	1	1	2					
94	35	2	2	2	2	2	2	2	1	2	1	1					
95	43.4	2	1	2	2	2	2	2	1	1	1	2					
96	23.4	2	1	2	2	2	1	1	1	1	1	2	3	1	0	1	1
97	46.7	2	2	2	2	2	2	2	3	2	2	2					
98	40	2	2	2	2					1	1	1					
99	20	2	2	2	2					1	1	2	3	3		4	2
100	29.9	2	1	2	2	2	2	1	1	2	1	2					
101	35	2	1	2	2	2	2	1	1	2	2	2	5	1	32.5	3	2
102	41.7	2	1	1	2	2	2	2	3	1	1	2	5	1	20	3	2
103	38.3	2	2	2	2					1	1	2	4	3		4	2
104	38.4	2	2	2	2	2	2	2	1	1	1	2					
105	56.6	2	1	2	2	2	2	2	1	1	1	2	5	1	22.5	1	1
106	46.7	2	1	1	2	2	1	1	1	1	1	2					
107	38.4	2	1	2	2	2	2	1	1	1	1	2	4	1	36.25	2	2
108	36.25	2	1	2	2	2	1	1	1	1	1	2	6	1	16.25	1	1
109	30	1	2	2	2	2	1	1	1	1	1	2	4	3		4	2
110	33.7	2	2	2	1	2	1	1	1	1	1	2	4	1	0	1	1
111	45	2	1	2	2					2	1	2					
112	23.3	2	2	2	2	2	1	1	1	1	1	2	3	1	0	1	1
113	26.6	2	2	2	2	1	1	1	1	1	1	2					
114	33.4	1	1	2	2	2	2	2	1	2	2	1	4	1	31.25	1	1
115	26.7	2	2	2	2	1	1	1	1	1	1	2					
116	45	1	1	1	2					1	1	2	5	3		4	1
117	43.75	1	2	2	2	2	1	2	2	1	1	2	4	1	16.25	1	1
118	37.5	1	1	1	2	2	2	2	1	1	1	2	3	1	0	1	1
119	31.7	1	2	2	2	2	2	2	2	1	1	2	3	3		4	2
120	23.3	2	1	1	2	2	1	1	1	1	1	2					

S.No.	NAME	AGE	Sex M-1, F-2	Affected sideU-1, B-2	Ear dischargeY-1, N-2	Duration(yr)	Last episode of discharge (days)	TinnitusY-1, N-2	NoseNil -1, Allergy-2	Comorbidities Y-1, N-2	Previous surgeryY-1, N-2	Perforation size S/M-1, L/ST-2	Middle ear mucosa D-1, M-2
121	VANMATHI -1	17	2	2	1	15	2	2	2	2	2	2	2
122	TAPOSH KUMAR DAS	43	1	1	1	8	4	2	1	2	1	2	1
123	ARAVIND KUMAR	20	1	2	1	10	1	1	1	2	2	2	2
124	SATHYA DEVI -1	27	2	1	1	2	7	2	2	2	2	2	2
125	PUSPALATHA	27	2	1	1	1	45	1	1	2	2	2	1
126	SURESH. S -1	21	1	1	1	0.5	180	2	1	2	2	2	1
127	SATHYA. C	23	2	1	1	2	1	1	1	2	2	2	2
128	HIMANSHU	35	1	2	1	20	3	1	1	2	2	2	2
129	RAGHUPATHY	35	1	2	2	N	N	2	1	2	2	2	1
130	GUNA .S	42	2	1	1	3	60	2	1	2	2	1	1
131	SHARMILA	24	2	2	1	15	90	2	1	2	2	2	2
132	BINOY KUMAR	37	1	2	1	12	15	2	1	2	2	2	2
133	SANTHOSH GOPE	24	1	2	1	18	365	2	1	2	2	2	2
134	VENKATESAN	59	1	1	1	7	90	2	1	2	2	2	1
135	AKRAMUL HAQUE SHEIK - 1	49	1	1	1	6	365	1	1	2	2	2	2
136	PARITHOSH GHOSH	43	1	2	1	10	90	2	1	2	2	2	1
137	KARPAGAM	54	2	1	1	7	7	1	1	2	2	1	2
138	MADAN KUMAR 1	18	1	2	1	16	45	2	1	2	2	2	1
139	MADAN KUMAR 1	18	1	2	1	16	45	2	1	2	2	2	1
140	GOPI .R	27	1	2	1	20	5475	2	1	2	2	2	1
141	YUVARAJ - RIGHT	23	1	2	1	20	30	1	2	2	2	2	1
142	LAKSHMI R	50	2	2	1	1	7	2	1	2	2	1	2
143	DEEPAK KUMAR	23	1	1	1	1.5	45	2	1	2	2	2	1
144	DILIP KUMAR SARKAR	50	1	2	1	1.5	90	2	1	2	2	2	1
145	NAMITA MONDAL	56	2	1	1	50	3	2	1	2	2	2	1
146	KALPANA S	32	2	1	1	30	7	2	1	2	2	2	2
147	SASIKALA S	27	2	1	1	1	365	2	1	2	1	2	2
148	MURUGAN B	34	1	1	1	10	30	2	1	2	2	2	1
149	BINA SINGH	37	2	1	1	15	1	1	1	2	2	2	2
150	CHIRANJIVI SHARMA	42	1	1	1	34	120	1	1	1	2	2	1
151	SNEHANSHU CHAKRABRTHY	40	1	1	1	14	270	1	1	2	1	2	2
152	SANTHOSH R	26	1	1	1	5	180	2	1	2	2	2	2
153	SABITA ROY	58	2	2	1	5	30	1	1	2	2	1	2
154	IBRAHIM ALI	39	1	1	1	15	30	1	1	2	2	2	1
155	PANKAJ KUMAR SAW	29	1	2	1	20	90	2	1	2	2	2	2
156	LALITHA SHARMA	57	2	1	1	1	30	2	1	2	2	2	1
157	MUTHULAKSHMI	45	2	1	1	0.58	1	1	1	2	2	1	2
158	SUSHIL SAMANTA	45	1	1	1	15	150	1	1	2	2	2	1
159	RAJARAJAN - 1	42	1	1	1	3	60	1	1	1	1	2	1
160	SWATHI - 1	17	2	1	1	2	150	2	1	2	2	1	1

S.No.	Avg AB gap R & L	SNHL Y-1, N-2	PreOPValsalvaP-1, NP-2	Pre OP I&D (P,P-1), (P,NP/NP,NP/NP,P-2)	Tympanic remanent (Tsp/ode)-1,N-2	Mastoid C-1, S-2	AntrumN-1, OE-2	AditusP-1, B-2	Aditus made P-1,PP-2,B-3	OssiclesN-1, (ER,TS-2),NA-3	N-1,(R/A-2),NA-3	Catlilage/boneY-1,N-2	Follow up(mnths)	Post OP graft (N,&GRN-1), CP-3	AB gapR&L	Tymps(A-1),(C-2),(B-3),(CP-4)	POST Valsalva P-1,Np-2
121	23.3	2	2	2	2	2	1	1	1	1	1	2					
122	38.4	1	2	2	2	2	1	1	1	1	1	2	3	3		4	2
123	35	2	1	1	2	2	2	2	1	1	1	2					
124	23.3	2	1	2	1	1	1	1	1	2	2	1					
125	32.5	2	2	1	2	2	1	1	1	1	1	2	3	1	0	1	2
126	15	2	1	1	2					1	1	2	3	1	0	1	1
127	40	2	1	2	1	2	2	2	1	2	1	2					
128	33.75	2	1	2	2	2	1	1	1	1	1	2					
129	10	2	1	1	2					1	1	2	3	1	0	2	1
130	39.75	1	1	1	2					1	1	2	3	1	41.4	1	1
131	12.5	2	1	1	2					1	1	2					
132	23.75	1	2	1	2	2	2	2	1	1	1	2					
133	40	2	1	2	2	2	2	2	2	1	1	2	3	1	0	1	1
134	16.25	1	1	1	2	2	2	2	1	1	1	2	4	3		4	1
135	26.25	2	1	1	2					1	1	2					
136	40	2	1	1	1					2	2	2	4	1	36.25	1	1
137	30	2	1	2	2	2	2	1	1	1	1	1	3	1	36.25	3	2
138	45	2	1	2	2	2	1	1	1	2	2	2	9	1	0	1	1
139	23.75	2	1	2	2	2	1	1	1	2	2	2	5	3		4	1
140	23.75	2	1	2	2	2	2	2	1	1	1	2	3	1	18.75	1	1
141	30	2	1	1	2	2	2	2	1	1	1	2	3	1	0	1	1
142	43.75	2	2	2	2	2	1	1	1	1	1	2					
143	15	2	2	1	2					1	1	2					
144	50	1	2	2	2	1	1	1	1	1	1	2					
145	35	1	1	2	2					1	1	2	3	3		4	1
146	42.5	2	1	1	2	2	2	2	2	2	2	2	3	3		4	2
147	17.5	2	2	1	2					1	1	2					
148	23.75	2	2	2	2	2	1	2	2	1	1	2	3	1	20	1	1
149	38.5	1	1	1	2	2	2	2	2	1	1	2					
150	21.25	2	2	2	2	1	1	1	1	1	1	2					
151	37.5	2	1	1	2	1	1	2	2	1	1	2					
152	17.5	2	1	2	2					1	1	2	3	1	13.75	3	1
153	18.75	2	2	1	1					1	1	2					
154	35	1	1	2	1	2	1	1	1	1	1	2					
155	31.25	2	2	2	2	2	2	1	1	1	1	2					
156	16.25	1	2	2	2					1	1	2	3	1	0	1	1
157	26.25	2	2	2	2	2	2	2	2	1	1	2	15	1	12.75	1	2
158	28.75	1	1	2	2	2	1	1	1	1	1	2					
159	28.75	1	1	1	2	2	2	2	2	1	1	2					
160	30	2	2	2	1					1	1	2					

S.No.	NAME	AGE	Sex M-1, F-2	Affected side U-1, B-2	Ear discharge Y-1, N-2	Duration(yr)	Last episode of discharge (days)	Tinnitus Y-1, N-2	Nose Nil -1, Allergy-2	Comorbidities Y-1, N-2	Previous surgery Y-1, N-2	Perforation size S/M-1, L/ST-2	Middle ear mucosa D-1, M-2
161	AMAR BARI LEFT	22	1	2	1	15	30	1	1	2	2	2	2
162	SAJJADALI MONDAL	41	1	1	1	7	150	1	1	2	1	2	1
163	VIVEKANANDAN	31	1	1	1	2	30	1	1	2	2	2	1
164	RAMANI -1	45	2	1	1	2	90	2	1	2	2	2	1
165	SELVAM R	41	1	1	1	4	60	2	1	2	2	1	1
166	ANJUMANARA AKTER BEAUTY	24	2	1	1	5	20	1	1	2	2	2	1
167	BISWAJIT BANIK	25	1	1	1	4	60	1	1	2	2	2	1
168	THAMILARASI	26	2	1	1	0.08	30	2	1	2	2	2	1
169	DHIVYA R	21	2	1	1	15	30	1	1	2	2	2	2
170	NAVEEN TAJ	17	2	2	1	10	1825	2	1	2	2	2	2
171	GOPAL CHAKRABORTHY	48	1	1	1	0.416	30	1	1	1	2	2	2
172	UTHIRAPATHY	21	1	1	1	10	30	2	1	2	2	2	1
173	MUSLIMA KHATUN	46	2	1	1	2	30	2	1	2	2	1	2
174	MITUNJOY GANGALY	28	1	1	1	15	14	2	1	2	2	2	2
175	SIVALINGAM L	19	1	2	1	5	60	2	2	2	2	1	1
176	RESHMA PERVEEN	28	2	1	1	5	180	2	1	2	2	2	1
177	MANORMANI	41	2	1	1	30	30	2	1	2	2	2	1
178	RANI	49	2	2	1	2	365	2	1	2	2	2	1
179	BHARATHIRAJA 1 R	32	1	2	1	1.5	45	1	1	2	2	1	1
180	NIRMALA K	20	2	2	1	10	30	2	1	2	2	2	2
181	SAKTHIVEL. C	17	1	2	1	10	7	2	1	2	2	2	1
182	FATHICK CHANDRA PRAMANIK L	41	1	2	1	35	60	2	1	2	2	2	2

S.No.	Avg AB gap R & L	SNHL Y-1, N-2	PreOPValsalva P-1, NP-2	Pre OP I&D (P,P-1), (P,NP/NP,NP/NP,P-2)	Tympanic remanent (Tsp/ode)-1,N-2	Mastoid C-1, S-2	AntrumN-1, OE-2	AditusP-1, B-2	Aditus made P-1,PP-2,B-3	OssiclesN-1, (ER,TS-2),NA-3	N-1,(R/A-2),NA-3	Catilage/boneY-1,N-2	Follow up(mnths)	Post OP graft (N,&GRN-1), CP-3	AB gapR&L	Tymps (A-1),(C-2),(B-3),(CP-4)	POST Valsalva P-1,Np-2
161	33.75	2	2	2	2	2	2	2	1	1	1	2	3	1	20	2	2
162	30	2	1	1	1	2	1	1	1	1	1	2					
163	22.5	2	1	2	2					1	1	2					
164	20	2	2	1	2					1	1	2					
165	26.25	1	2	2	2					1	1	2					
166	48.75	2	2	2	2	2	2	2	2	1	2	2					
167	21.25	2	1	2	1					1	1	2					
168	42.5	2	1	1	2					1	1	2					
169	53.75	1	2	2	2	2	2	1	1	1	1	2					
170	47.5	2	2	2	1					1	1	2	3	1	43.75	3	2
171	28.75	1	2	2	2					1	1	2					
172	45	2	1	2	1					1	1	2					
173	22.25	1	2	1	2	1	2	2	1	2	2	1	3	1	31.25	2	2
174	42.5	2	1	2	2	2	2	2	1	1	1	2					
175	0	2	1	2	2	2	1	1	1	1	1	1					
176	47.5	2	1	1	2					1	1	2					
177	52.5	2	1	1	2					1	1	2	3	1	28.75	1	2
178	26.25	2	1	1	1					1	1	2	3	3		4	1
179	30	2	1	2	1					1	1	2					
180	36.25	2	1	1	2	2	1	1	1	1	1	2	2	1	22.5	1	1
181	26.75	2	1	2	2	2	2	2	2	1	1	2					
182	25	2	1	2	2	2	1	1	1	1	1	2					